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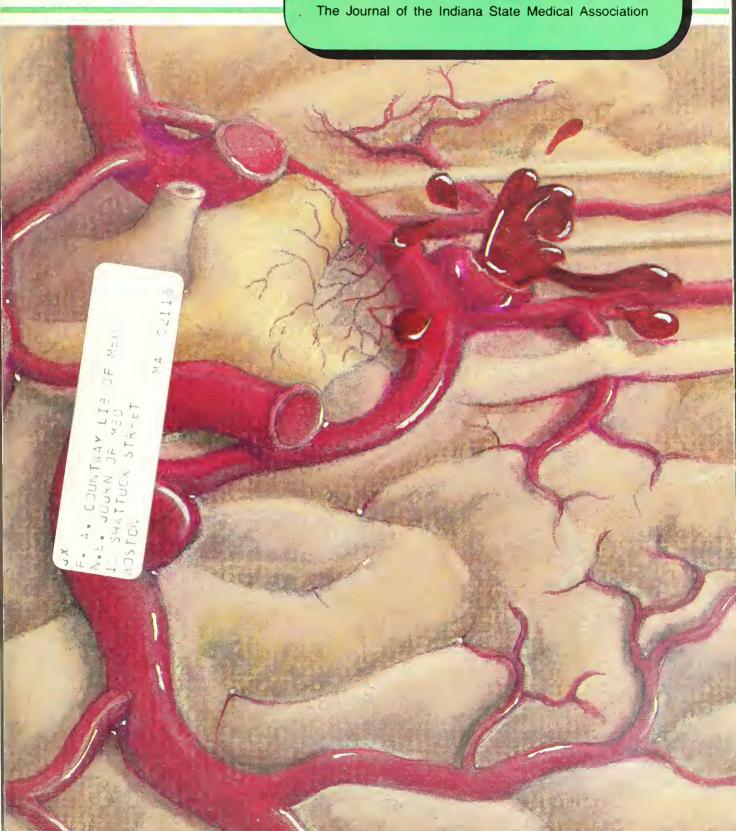
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INDIANA MEDICINE

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Vol. 78, No. 5 MAY 1985

Devoted to the interests of the medical profession and public health in Indiana since 1908.

SCIENTIFIC ARTICLES DEPARTMENTS, MISCELLANEOUS Neurologic Signs and Symptoms Related to CME: Polycythemia in OTC Diet Pills..... 388 Percutaneous Renal Stone Future File. 358 PD CRITICAL CARE: Extraction: Experience with Parental Stress during and Public Health Notes 360 393 One Stage Procedures after Pediatric ICU Cancer Corner. 365 FEATURES Editorials.... 416 CRITICAL CARE MEDICINE: Ethics and Medicine: Constitutional Amendment. 416 Management of Vasospasm 1. Introduction.... 403 417 from Ruptured Intracranial Auxiliary Report Notes from the Royal .420 Aneurysms with Induced College of Surgeons. 406 Book Reviews.... 422 News Notes.. 426 Commentary: Skin Diseases: Current Are Angioplasters Split? . 410 Concepts and Therapy CME Awards 429 1. Acne.....378 New ISMA Members 429 President's Message: Cardiac Transplantation: Advances in Medicine 412 CME Quiz.... 437 Results of a Two-Year ISMA's Leadership. 438 Experience Obituaries. 439

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Our cover depicts a ruptured anterior communicating artery aneurysm which is spilling blood into the spinal fluid. The disease has a mortality rate as high as 50%. The patient will complain of an extremely intense headache, which began after "something exploded in my head." Dr. Terry G. Horner. an Indianapolis neurosurgeon, discusses the critical care management of this problem, beginning on page 376.—DRAWING BY BRENDA KESTER, MEDICAL MEDIA PRODUCTIONS, METHODIST HOSPITAL OF INDIANA

MUSEUM NOTES

CHARLES A. BONSETT, M.D., Indianapolis



The Indiana State Medical Society (ISMS), at least in the number of new members. At that year's annual meeting, 34 new applicants were elected to add to the 97 physicians then constituting the organization.

Total income for the year 1852 had been \$82, which when added to the \$23.29 balance from 1851, made a grand total of \$105.29. After expenses (publishing and mailing the *Transactions of the Indiana State Medical Society*) a total of \$25.75 remained in the hands of the treasurer as of May 1, 1853, so things were looking up.

The annual meeting at that time was held in May, not in October as at present. And, except for the annual mailing of the *Transactions*, there was no notification except through local newspapers of the date and place of the meeting. The *Indianapolis Locomotive* carried the following ad, for example, on page 3 of the May 14, 1853 edition: "Indiana State Medical Society — The Fourth Annual Meeting of the State Medical Society will be held in Lafayette on Wednesday, May 18, 1853. Papers friendly to the profession will please notice the fact."

That someone might be "unfriendly" to the profession is suggested by the ad immediately above the one just mentioned: "Medical Notice—The Third Annual Meeting of the Botanic Society of Indiana will meet in Indianapolis on Tuesday, 24th of May, 1853. All true friends of Medical Reform are invited to attend."

The names of several members of the ISMS are found in this edition of the paper, e.g., the ad of Dr. John Bobbs on page one: "John S. Bobbs, Surgeon, Office on Market Street, on the second square east of the Court House, Indianapolis, Indiana."

The reform motives of the "botanics" were apparently of no concern to the "regulars" whose meeting commenced promptly at 9 a.m. in the Court House at Lafayette on the appointed day.



The Old Lafayette Court House Is Gone, But the ISMS Meeting Held There in 1853 Is Worth Recalling . . .

Shown on this page is that court house, which was razed in 1876 to make way for the structure that now occupies the square. Physicians living at a distance from Lafayette would have made the journey by train. Calvin Fletcher, in his diary, indicates that the trip from Indianapolis at that time could take as long as five hours.

Dr. John Bobbs started the proceedings with a motion that Drs. Grimes and Bray conduct the president to the chair. Dr. Jeremiah H. Brower (1798-1866), a founding member of the Society in 1849 and a frequent contributor to the *Transactions*, was the president for 1853. His first order of business was the election of the 34 new candidates to membership.

Most interesting of these candidates at this late date is Dr. William W. Mayo. Dr. Mayo would later move on to Minnesota and establish St. Mary's Hospital, which would ultimately develop into the Mayo Clinic. At this particular time, he was a young Hoosier physician in practice at Lafayette with his preceptor, Dr. Elizur H. Deming.

Dr. Mayo was born near Manchester

in England in 1819. His early training in England was directed toward medicine. He came to New York in 1845 and spent two years as a chemist. He was in Lafayette by 1847, where he earned his living as a tailor. Together with a partner, Alphonso Roath, he opened a "House of Fashion" on Main Street, one door west of Third Street. Just how and when he met Dr. Elizur Deming is not clear.

At any rate, it was through the influence of Deming, who had been practicing in Lafayette since 1834 and who had been a faculty member of the LaPorte Medical College since 1847, that Mayo again turned his attention to medicine. Dr. Deming served as his preceptor, and during the winter of 1849-50 Mayo attended a session of lectures at the LaPorte school and was awarded the M.D. degree on Feb. 14, 1850. Dr. Deming and Dr. Mayo later practiced at an office at the corner of Columbia and Wabash (Second) Streets in Lafayette.

On the occasion of the Fourth Annual ISMS Meeting in 1853, Dr. Mayo was in his 34th year. Dr. Deming (1797-1855) and Dr. Brower were both in there mid-fifties. Dr. Brower, on the second and final day of the 1853 meeting, assigned topics to certain physicians, to be reported to the membership at the next annual meeting. To the chemist, Dr. William Mayo, the topic of "Pathological Indications of the Urine" was given. (This was presented the following year and published in the 1854 *Transactions.*)

Dr. Deming was elected president to preside at the Fifth Annual Meeting, which would be held in Evansville on the third Wednesday in May 1854.

Dr. Brower's contribution to the 1853 session, in addition to his presidential address, was a report on vital statistics. He urged legislation to require the registration of marriages, births and deaths throughout the state. (More than a quarter of a century would pass before such laws would be enacted.)

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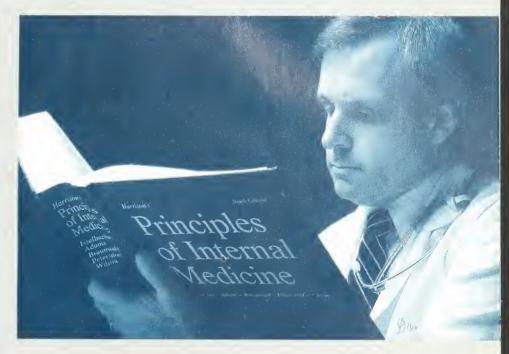
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WHAT'S NEW?

Geigy Pharmaceuticals has FDA approval for marketing Lopressor HCT^{**}, a combination of the beta blocker and hydrochlorothiazide. Tablets are in three sizes – 50/25 (containing 50 mgs of metoprolol tartrate and 25 mgs of hydrochlorothiazide), 100/25 and 100/50. Dosage once a day will often be sufficient.

3M has a new hip system. 209 26mm Hip System is a versatile, cost-effective hip replacement system named for the DRG number for hips. The one-size system allows a "mix and match" approach to component selection that can reduce inventory. The surgeon can also choose to substitute a less expensive component within the system if it is appropriate for a patient.

Hewlett-Packard has five sizes of individual pressure cuffs and two pressure-cuff kits for use with HP 78354A patient monitor. Correct cuff size is essential for recording accurate readings. This family of cuffs is designed to meet all non-invasive blood-pressure monitoring situations.

Roche is introducing Coactin®, (amdinocillin/Roche), the first penicillin that binds with PBP-2. The binding allows the product to enhance antibacterial activity when used with other penicillins and cephalosporins. Coactin is indicated for treatment of urinary tract infections caused by susceptible strains of *E. coli, Klebsiella pneumoniae, Klebsiella* species and *Enterobacter* species, and bacteremia associated with severe urinary tract infections due to *E. coli*. It is available in 1 gram and 500 mg vials.

Abbott Laboratories has FDA approval to market a diagnostic test to screen blood and blood products for evidence of the virus believed to be the cause of AIDS. The new test is called "Abbott HTLV-III EIA." The test may be performed on the Abbott Quantum and on other members of the Quantum line, all of which are in plentiful supply worldwide.

Lanier Business Products is unveiling a new medical office software package. The new software, called Practi-CareTM Medical System, is being introduced through an 11-city kiekoff before a slated mid-year national release. Practi-Care handles all required administrative tasks in the physician's office. Among them: billing, insurance claim submission, financial information reporting, practice analysis, recall notices, appointment scheduling, etc.

Schering has received authorization from the FDA to remove the time restriction from Diprolene Ointment. This potent topical anti-inflammatory corticosteroid was introduced in 1983 with a limitation of use to 14 days. With removal of the 14-day restriction is a recommendation that it be applied sparingly twice daily, and in amounts no greater than 45 grams per week. The ointment also should not be used underneath an occlusive dressing.

Kodak has a new dry-chemistry slide for its Kodak Ektachem analyzer for simultaneous determinations of unconjugated and conjugated bilirubin. Ektachem 400 and 700 analyzers also have added phosphorus to the tests available for both instruments.

Ross Laboratories announces TWO CAL™ HN High Nitrogen Liquid Nutrition, a complete, balanced, high-density enteral formula. It has a caloric density of 2 calories per mL. It contains 100% of the US RDA for vitamins and minerals in 1,900 calories (950 mL). It may be tube-fed or taken orally.

News of what is new in the medical supply industry is composed of abstracts from news releases by book publishers and manufacturers of pharmaceuticals, clinical laboratory supplies, instruments and surgical appliances. Each item is published as news and does not necessarily constitute an endorsement of a product or recommendation for its use by INDIANA MEDICINE or by the Indiana State Medical Association.

Roche has introduced a new antibiotic, Rocephin (ceftriaxome sodium/Roche). It is an injectable broad-spectrum cephalosporin that combines excellent antibacterial activity with an extended half-life. It is active against a wide range of grampositive and gram-negative bacteria. A single 1 gram dose produces serum levels over a 24-hour period that exceed inhibitory levels for 90% of Enterobacteriaceae, Haemophilus influencae, Streptococcus pneumoniae, and Staphylococcus species, except those resistant to methicillin.

Precision Dynamics has a new Franzen Needle Guide for use in obtaining needle biopsy of the prostate by the transrectal approach. The guide and accompanying special Luer needles are used in combination with Precision's Cameco Syringe Pistol, which creates the vacuum necessary to withdraw biopsy specimens through the needle.

UroTec Systems Corporation is introducing a catheter that permits higher flow rates, has greater resistance to encrustation and a number of benefits not shared by latex and Teflon-coated catheters. It is known as "UroTec/Franklin Simplastic Catheter." It is made of a new high-quality polyvinylchloride and can achieve good drainage with smaller sizes, which are much better tolerated by the urethra.

Modern Healthcare Concepts is introducing the "Care Chair." The frame of the chair is constructed with metalreinforced P.V.C. and fitted with Naugahyde cushions which can be removed to convert the chair for use in a shower bath. Another feature is a seat that is fashioned to allow the patient to be transported to a position over the toilet. The chair straddles the toilet or will admit a plastic wastebasket when a toilet is not immediately available. Only one attendant is needed for such a function.

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FUTURE FILE

Nutrition and Aging

"Nutrition and Aging" is the topic of the fifth annual Bristol Myers Symposium on Nutrition Research, which will meet Oct. 31 and Nov. 1 at the Copley Plaza Hotel in Boston.

The symposium is part of a program of nearly \$2.5 million in unrestricted grants for nutritious research at 13 in stitutions that Bristol-Myers and its Mead Johnson & Co. subsidiary introduced in 1980.

For registration information, contact Ralph D. Weaver, Bristol-Myers Co., 345 Park Ave., Room 43-38, New York, N.Y. 10154 – (212) 546-4319.

Technology Assessment

"Medical Technology Assessment for Health Professionals" is the subject of a week-long course in health policy and management that will be offered by the faculty of the Sloan School of Management of MIT. The 1985 MIT summer session, June 17-21, will feature a distinguished visiting faculty.

CME credit is being offered by Tufts University School of Medicine.

Among the topics will be thirdparty payment for medical practices, medical/scientific assessment of emerging technologies, communication among medical technology professionals, and health research and development.

Contact the Director of the Summer Session, Room E19-356, Massachusetts Institute of Technology, Cambridge, Mass. 02139.

Reflux Esophagitis

"Medical and Surgical Review of Reflux Esophagitis and the Angelchik Prosthesis" will be discussed June 14-15 at the Concourse Hotel, Madison, Wisc.

The review is for physicians, including primary care providers. AMA Category 1 credit is 11 hours.

Contact Sarah Z. Aslakson, 465B WARF Bldg., 610 Walnut St., Madison, Wise. 53705 – (608) 263-2856.

Hand Surgery Courses

The American Society for Surgery of the Hand is sponsoring four courses for hand surgeons:

May 9·11 = "Vascular Disease and In juries in the Upper Limb" will be addressed by a multi-disciplinary group of specialists at the Hyatt Regency, Baltimore.

May 13-15—"Primary Care of Hand Injuries" will be discussed by a distinguished faculty at the Clinic Inn in Cleveland, Ohio.

June 19-22—"Common Problems and Difficult Decisions in Hand Surgery" will be covered by a 3½-day course at the Niagara Hilton, Niagara Falls, N.Y.

Sept. 16-18—"Trauma to the Hand: Diagnosis, Treatment and Functional Restoration" will be discussed for the benefit of physicians who have periodic responsibility for trauma, at the Campus Inn, Towsley Center, Ann Arbor, Mich.

Contact the Society at 3025 S. Parker Road, Suite 65, Aurora, Colo. 80014 – (303) 755-4588.

Child Abuse

"The Seventh National Conference on Child Abuse and Neglect" will be held Nov. 10-13 at the Chicago Hilton and Towers.

It is sponsored by the National Center on Child Abuse and Neglect, U.S. Dept. of Health and Human Services.

Convention program details and registration packets are available from Seventh National Conference Hqs, c/o Moorevents, Inc., 400 N. Michigan Ave., Suite 2300, Chicago 60611 – (312) 644-5997.

The Journal of the American Medical Association publishes a list of CME courses for the United States twice yearly. The January listing features courses offered from March through August; the July listing features courses offered from September through February.

Optimal Cine Imaging

"Principles of Optimal Cine Imaging" is the subject of an education seminar offered by Vari-X, Inc.

Although seminars will be conducted in several cities, the closest will be one held in Chicago Sept. 7 and 8. Such topies as optimizing the equipment system, processor quality control techniques, and evaluation and selection of cine films will be covered.

For more information, contact Vari-X, Inc., 17601 Fitch, Irvine, Calif. 92714-(800) 421-6841.

Indiana University CME

For the Primary Care Physician

May 15 – Diagnosis & Management of Cardiac Arrhythmias, Indianapolis.

May 21-23 — Family Practice Update — Part I, Indianapolis.

June 1-Diabetes Update, Indianapolis.

June 5-Richter Child Psychiatry Conference, Indianapolis.

June 5—Treatment of Hematologic Malignancies, Indianapolis.

July 23-25 – Family Practice Update – Part II, Indianapolis.

For the Specialist

May 29-Neonatal Perinatal Medicine, Merrillville.

July 8-17 - Anatomy & Histopathology of the Head & Neck and Temporal Bone, Indianapolis.

July 26-27 – Management of the Patient with Breast Cancer, Indianapolis.

For additional information, contact the CME Division, Indiana University School of Medicine – (317) 264-8353.

Geriatrics Meeting

The 42nd annual meeting of the American Geriatrics Society and the sixth annual meeting of the American Federation for Aging Research will be conducted July 11 and 12 at the Sheraton Centre in New York City.

For registration details, contact the Society at 10 Columbus Circle, Suite 1470, New York, N.Y. 10019 – (212) 582-1333.

PD Respiratory Disease

A "Neonatal and Pediatric Respiratory Disease Conference" will be conducted June 6-9 at the Sheraton-Sand Key Resort Hotel, 1160 Gulf Blvd., Clearwater Beach, Fla.

For the program and details, contact Myrtle E. Larson, R.N., Gulf Coast Lung Assn., 6160 Central Ave., St. Petersburg, Fla. 33707 – (813) 347-6133.

Emergency Medicine

The American College of Emergency Physicians, California Chapter, announces its 14th annual scientific assembly June 7 to 9 at the Doubletree Hotel, Monterey, Calif.

Tuition is free. There will be fee courses offered in addition to the free scientific assembly.

For a brochure, contact CAL/ACEP, 505 N. Sepulveda Blvd., Manhattan Beach, Calif. 90266—(213) 374-4039.

Sports Medicine

The American College of Sports Medicine will hold its 1985 annual meeting May 26 to 29 at the Opryland Hotel in Nashville, Tenn.

The event is the largest annual scientific sports medicine session in the world. More than 500 scientific papers will be presented, along with 13 half-day symposia. Registration is \$85 for members, \$110 for non-members.

For further information, call Jane C. Shepard, Director of Meetings, at (317) 637-9200.

Wilderness Medicine

"Wilderness Medicine" will be the subject of a CME conference to be conducted by the UC San Diego School of Medicine at Lake Tahoe, Nev., Aug. 12 to 16. It carries 23 hours of Category 1 credit.

Contact the Office of CME, M-017, UC San Diego School of Medicine, La Jolla, Calif. 92093—(619) 452-3940.

Emergency Care Problems

The fifth annual Common Emergency Care Problems Program will be conducted July 17 and 18 at the Sheraton Inn and Conference Center, Madison, Wisc. It is accredited for 12 AMA Category 1 hours.

Contact Sarah Z. Aslakson, 465B WARF Bldg., 610 Walnut St., Madison, Wisc. 53705 – (608) 263-2856.

Pediatrics Symposium

"Pediatrics for the Practicing Physician" will be the subject of a symposium sponsored by the Medical College of Ohio affiliated hospitals and two Ohio pediatric societies on Sept. 20 and 21 at the Sheraton Westgate, Toledo.

The fee for physicians is \$160, residents \$70, and nurses \$60.

Write to Robert C. Bobo, M.D., St. Vincent Medical Center, 2213 Cherry St., Toledo, Ohio 43608.



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PUBLIC HEALTH NOTES

The Acquired Immune Deficiency Syndrome (AIDS) has been described by health professionals as one of the most serious epidemics confronting man in modern time. Through mid-March, 1985, about 9,000 cases had been reported nationwide, and it has recently been estimated that there will be 40,000 new cases in the next two years. It has also been estimated that at least 400,000 persons have antibodies to HTLV-III virus, indicating current or past infection.

Approximately 73% of cases are in male homosexuals, 17% in I.V. drug users, 4% in Haitians, and 2% in persons who have hemophilia or who have received blood transfusions or blood products. A number of cases also have been reported in infants who were infected pre- or perinatally. The two-year mortality rate exceeds 70%, and one study of AIDS patients showed that they have an average life span of 224 days after being hospitalized for their first opportunistic infection.

Since 1981, 35 cases of AIDS have been reported in Indiana and 19 of these persons have died.

AIDS appears to be caused by infection with Human T-Cell Lymphotropic Virus Type III (HTLV-III). The virus has been recovered from blood, seminal fluid, and saliva of patients with AIDS. It seems to show selective tropism for cells of the helper-inducer lymphocyte subset, in which it induces a cytopathic effect with resultant deficiency in immune system functioning.

HTLV-III infection can result in a broad spectrum of clinical conditions ranging from asymptomatic infection to full-blown AIDS. Between these extremes is a condition of persistent lymphadenopathy (the chronic lymphadenopathy syndrome), as well as a syndrome characterized by minor conditions clinically associated with immunosuppression (the AIDS-related complex). Some individuals with the chronic lymphadenopathy syndrome and the AIDS-related complex go on to

develop full-blown AIDS, but at present it is unclear what percentage this will be.

There is much variation in the clinical picture that leads to the diagnosis of AIDS. Some patients have no specific symptoms or complaints until they develop symptoms and signs of

40,000 New Cases
of AIDS Expected
in Next Two Years

one of the specific opportunistic infections or malignancies associated with AIDS. Others may have variable periods of nonspecific problems such as weakness, malaise, weight loss, diarrhea, and/or generalized lymphadenopathy preceding the onset of the opportunistic infection or malignancy that allows the diagnosis of AIDS to be made. There continues to be no effective treatment which will allow restoration of normal immune functioning. Work continues on developing a vaccine, but this is proving to be a very difficult task.

Recently, a test for antibodies to HTLV-III was approved by the FDA. This test will be used by blood and plasma centers to screen donated blood for evidence of infectiousness. However, there also will be persons at risk for developing AIDS who will want the test because they believe it will give them information about their own condition.

The availability of the test raises three important issues. First, the test has serious limitations. A positive test only means that a person was probably infected at some undetermined point in the past with the virus. It does not allow one to determine if the person is currently infected, if he will develop AIDS, or if he is infectious to others. A negative test cannot entirely rule out the possibility that the individual is currently infected. These things should be very carefully explained to any high risk person who wishes to be tested, and such an explanation may well result in many of these individuals then choosing not to have the test.

The State Board of Health believes that persons should not use the test in an attempt to try to learn about their own condition because the test simply will not provide the answers.

A second issue here is that high risk persons, some of whom are infected and have virus in their blood, should not donate blood at blood or plasma centers in order to have the test. Rather, they should see a physician if they are interested in being tested. However, since not all high risk persons will be willing to contact a physician, so-called alternative collection sites will be set up in several communities around the state where the individuals can learn more about the test and where, if they still wish to be tested, they can submit specimens.

A third issue is that a very small percentage (perhaps less than 1%) of blood donors will have a positive antibody test. In some, this will represent a false-positive, but all these persons will need evaluation by a physician.

In summary, AIDS is a serious problem with a rapidly expanding number of cases being reported. Physicians need to become knowledgeable about AIDS and the HTLV-III antibody test for several reasons. First, they may see previously undiagnosed persons who have AIDS. And this can occur in small communities as well as in large metropolitan areas. Second, they may be contacted by high risk individuals who are interested in being tested, or who have had a positive antibody test

CONTINUED ON PAGE 386

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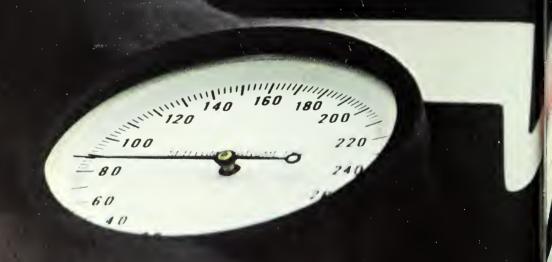
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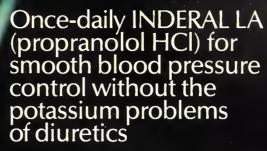
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Once-daily For beta-1/beta-2 NDERAL LA (PROPRANOLOL HCI) LONG ACTI

BRIFF SUMMARY (FOR FULL PRESCRIBING INFORMATION SEE PACKAGE CIRCULAR)
INDERAL* LA brand of propranolol hydrochlorid+ (Long Acting Capsules)
DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg. 120 mg. and 160 mg capsules
CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor situating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL. The chronotropic inotropic and vasodilator responses to beta adrenergic stimulation are decreased proportionately. INDERAL LA Capsules (80 120, and 160 mg) release propranolol HCI at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA Occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period hie areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline

hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to fNDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect. INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of reini release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum polassium concentration when used in the treatment of hypertensive patients.

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period. The net physiol INDERAL LA should not be considered a simple mg for mg substitute for conventional

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated

the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache
efficacy of propranoiol in the treatment of a migraine attack that has started has not been
ableted and propragoly is not indicated for such use. The el

The efficacy of progranolol in the treatment of a migraine attack that has started has not been established and progranolol is not indicitated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of progranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock. 2) sinus bradycardia and greater than first degree block. 3) bronchial asthma, 4) congestive heart tailure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with iNDERAL.

INDERAL WARNINGS. CARDIAC FAILURE. Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart purchase.

muscle
IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE continued use of beta blockers
can in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart
failure, the patient should be digitalized and/or treated with diuretics, and the response
observed closely or INDERAL should be discontinued (gradually, if possible)

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atheroscierotic heart disease who are given proprianolol for other indications.

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodila-

tion produced by endogenous and exogenous catecholamine stimulation of beta receptors MAJOR SURGERY. The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthe sia and surgical procedures.



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INDERAL (propranolol HC) like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e.g. dobutamine or isoproterenol. However such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

i blockers DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the ap-

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin. THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore abrupti withdrawal of propranoiol may be followed by an exacerbation of symptoms of hyperthyroidism including thyroid storm. Propranolol does not distort thyroid function tests in PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME several cases have been reported in which after propranoiol. The tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case, this resulted after an initial dose of 5 mg propranoiol.

proprantial PRECAUTIONS. General Proprantial should be used with caution in patients with impaired hepatic or renal function. INDERAL (proprantial HCI) is not indicated for the treatment of hepatic or renal function I hypertensive emergencies

hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be fold that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

Clinical Laboratory Tests. Elevated blood urea levels in patients with severe heart disease elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS. Patients receiving catecholamine-depleting drugs such as reservine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

hypotension

Carcinogenesis, Mulagenesis, Impairment of Fertility. Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mince, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was

levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug. Pregnancy. Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose. There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential insk to the fetus. Nursing Mothers. INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman. Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have required the withdrawal of therapy. Cardiovascular bradycardia, congestive heart failure, intensification of AV block hypotension, paresthesia of hands, thrombocytopenic purpura.

Raynaud type

Central Nervous System lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatomia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal nausea vomiting, epigastric distress abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat laryngospasm and respiratory distress.

Respiratory bronchospasm.

Hematologic agranulocytosis nonthrombocytopenic purpura, thrombocytopenic

purpura
Auto-Immune In extremely rare instances, systemic lupus erythematosus has been

Miscellaneous alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol)

Involving the skin serous membranes and conjunctivae reported for a beta blocker (practotol) have not been associated with propranoiol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranoiol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL labiets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels. Relitation may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval HYPERTENSION—Dosage must be individualized. The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily of higher until adequate blood pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—Dosage must be individualized. Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained. Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily in angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued reduce dosage gradually over a period of a few weeks.

MIGRAINE Dosage must be individualized. The initial oral dose is 80 mg INDERAL LA nonce daily. The usual effective dose may read the top the start the description of the proper day to the proper day to the start. The dosage must be individualized. The initial oral dose is 80 mg INDERAL LA nonce daily. The usual effective dose never the 20 mg once daily.

(see WARNINGS)

MIGRAINE Dosage must be individualized. The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response is no obtained within four to six weeks after reaching the maximum dose. INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of

several weeks
HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily
PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

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CANCER CORNER

WILLIAM M. DUGAN, JR., M.D., INDIANAPOLIS





Camp Little Red Door

A special birthday will be celebrated June 9 to 15. That's when the fifth Camp Little Red Door will be held at Bradford Woods, Indiana University's Outdoor Education Center, located on State Road 67 north of Martinsville.

Camp Little Red Door is the only summer camp in Indiana that is exclusively for young cancer patients, ages 8-18. It is funded by the Little Red Door, Marion County Cancer Society, with help from generous individuals and civic organizations, such as the Rotary Foundation of Indianapolis, Inc., the Hook Drug Foundation, the Sertoma Club of North Indianapolis, and the American Legion Post 495.

Children and teen-age cancer patients have the same needs as other young people, and the camp adds a normalcy to their lives. It gives them the opportunity to develop new skills, make new friends and explore the great outdoors—all in the company of others like themselves.

More and more children are living with cancer, and its effect on their development must be minimized. Comments from the campers and their parents indicate that children leave the camp better equipped to deal with the impact of their illness on their lives.

Virginia M. Wagner, M.D., serves as chairman of the Little Red Door Summer Camp Committee. Under her direction, physicians and nurses from the James Whitcomb Riley Hospital for Children volunteer their time during the camp session.

Camp Little Red Door is open to young people in all stages of their illness and treatments. Bradford Woods has a well equipped infirmary that enables the camp physicians to handle all types of medical situations. Intravenous chemotherapy can even be given, if needed.

The fee per camper is only \$75.00, even though the actual cost is \$300.00 for each child who attends. Parents can arrange payment plans, and financial assistance is available. Space is available for 50 campers. (This year's reservation deadline was April 25.)

Children who attend can look forward to activities such as swimming, hiking, canoeing, fishing, cookouts, nature study, and overnight campouts.

Brochures and applications can be obtained by calling 317-925-5595 or by writing to Camp Little Red Door, 1801 N. Meridian Street, Indianapolis 46202.









May 1985

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To obtain Category 1 credit for this month's article, complete the quiz on page 437.



Polycythemia in the Newborn Infant

D. WADE CLAPP, M.D. JAMES A. LEMONS, M.D. RICHARD L. SCHREINER, M.D. Indianapolis Prelatively common in the neonatal period, occurring in 1 to 5% of all newborn infants. Clinical signs attributable to polycythemia/hyperviscosity are frequently subtle and nonspecific. In fact, the majority of infants with this entity are entirely asymptomatic and appear to have normal development. However, in some instances polycythemia/hyperviscosity may result in significant morbidity for an infant.

This paper will review the incidence, etiology, clinical presentation, treatment and outcome of infants with polycythemia and hyperviscosity. A brief discussion of the relationship between polycythemia and hyperviscosity will also be presented.

Relationship Between Polycythemia and Hyperviscosity

Most clinical signs of polycythemia are thought to be secondary to hy perviscosity. Viscosity is defined as the resistance to flow of one "layer" of a fluid over another layer. This is analogous to the concept of friction between two surfaces. Viscosity of blood is determined by three factors: the hematocrit, the deformability of the red blood cells, and plasma viscosity. The hematocrit is the most important of these factors.¹

Shown in Figure 1 is an important relationship between hematocrit and viscosity. Below a hematocrit of approximately 63%, an increase in the hematocrit produces a near linear increase in the viscosity of blood. However, above approximately 63% an increase in the hematocrit produces an exponential increase in the viscosity. Therefore, polycythemia with as sociated hyperviscosity in the neonatal period is generally defined as a hematocrit greater than 63-65%. This will be discussed in more detail later.

Etiology

The etiology of polycythemia may be divided into two major groups: (1)

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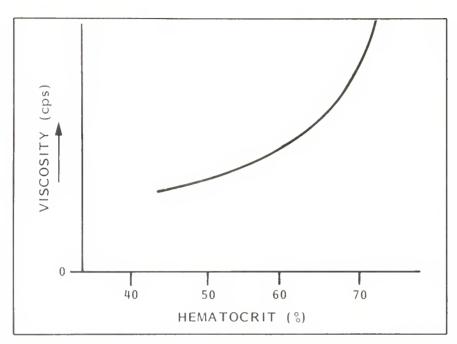


FIGURE 1: Effect of hematoerit on viscosity (cps, centipoise) in vitro, showing the increased viscosity with increasing hematocrit, especially when the hematocrit is greater than approximately 63%.

the fetus makes an excessive number of red blood cells (active); (2) the fetus receives a red blood cell transfusion (passive). A summary of the conditions which predispose newborn infants to polycythemia is shown in *Table 1*.

Active: Chronic fetal hypoxia is believed to be a major stimulus to fetal erythropoiesis. Factors which interfere with delivery of adequate oxygen to the fetus may therefore result in increased erythropoiesis and, eventually, polycythemia. The amount of oxygen delivered to the fetus is determined by the following parameters: uterine and placental blood flows, oxygen carrying capacity, partial pressure of oxygen and carbon dioxide, and oxygen diffusibility.

Polycythemia is known to occur in approximately 15% of all small for gestational age (SGA) infants. Many anatomic abnormalities have been described in the placenta of the SGA infant, including gross and microscopic infarctions, which reduce placental surface area available for

oxygen exchange. Maternal vascular disease is also associated with intrauterine growth retardation and, although not proven in humans, it is believed that a chronic reduction of uteroplacental blood flow is the cause of impaired fetal growth. This suboptimal blood flow in the SGA fetus is believed to cause mild chronic fetal hypoxia with a resultant physiological increase in erythropoietin activity and red cell production. Data to support this hypothesis include elevated erythropoietin levels in polycythemic, intrauterine growth retarded lambs and elevated erythropoietin levels in newborn polycythemic intrauterine growth retarded infants.1

Maternal cigarette smoking may cause an elevation in the carbon monoxide in maternal and umbilical cord blood as well as decreased uterine blood flow. Both of these factors reduce the oxygen carrying capacity of fetal blood and may increase erythropoiesis. In one study, the newborn infants of mothers who smoked had elevated hematocrits compared to in-

fants of mothers who did not smoke.5

Infants of diabetic mothers and other infants with hyperinsulinemia (e.g., Beckwith's syndrome) also demonstrate an increased incidence of polycythemia. Hyperinsulinemia in the fetus of the diabetic mother is associated with increased fetal plasma concentrations of erythropoietin.⁶

Other conditions in which intrauterine fetal erythropoiesis appears to be responsible for polycythemia include the chromosomal trisomies (13, 18 and 21), congenital adrenal hyperplasia and congenital thyrotoxicosis. A summary of the hypothetical physiological mechanisms by which many of the above disorders may result in fetal hypoxemia and subsequent polycythemia is presented in *Figure 2*.

Passive: The "passive" group of disorders that may result in polycythemia is less common than "active" causes and include: delayed cord clamping, maternal-fetal transfusion and twin-to-twin transfusion. Delayed clamping of the umbilical cord has been shown to increase the hematocrit by greater than 10%.7 This elevated hematocrit may persist for at least five days. Rausen et al estimate that significant twin-to-twin transfu-

TABLE 1 Etiologies of Polycythemia

Increased erythropoiesis (active)
Small for gestational age
Maternal vascular disease
Maternal hypoxemia
Maternal cigarette smoking
Maternal diabetes
Fetal hyperinsulinism
Beckwith's syndrome
Trisomies 13, 18, 21
Congenital adrenal
hyperplasia
Congenital thyrotoxicosis

Transfusion (passive)
Delayed cord clamping
Maternal-fetal transfusion
Twin-to-twin transfusion

sion occurs in approximately 15% of monochorial twins via vascular anastomoses (usually artery to vein).

Clinical Signs

Most of the clinical signs attributed to polycythemia are believed to be secondary to the elevated red blood cell mass in association with impaired organ perfusion. The most consistent findings include: lethargy, poor feeding with inadequate sucking, plethora, jitteriness, jaundice, tachypnea and peripheral cyanosis. There have also been case reports of congestive heart failure, testicular infarction, acute renal failure, peripheral gangrene, cerebral infarction and necrotizing enterocolitis.

Several laboratory abnormalities have been described in polycythemic infants, the most frequent of which is hypoglycemia.8 In some studies, more than 25% of infants with polycythemia have been hypoglycemic in the newborn period. However, it must be noted that screening glucose methods which rely upon a reagent strip tend to underestimate serum glucose concentrations because they measure whole blood glucose. As the hematocrit increases, the discrepancy between the estimate of the whole blood glucose concentration with the reagent strip and the plasma glucose concentration becomes greater. Abnormalities of the coagulation system have also been described, including circulating fibrin monomers, increased intravascular thromboplastin activity and transient thrombocytopenia. Hypocalcemia also occurs with greater frequency in polycythemic infants.

Radiological findings in polycythemic infants include pulmonary vascular congestion, pleural effusions and cardiomegaly.9

Diagnosis

The incidence of polycythemia varies according to the definition accepted, the site of blood sampling, 10 the time of sampling 11 and the timing

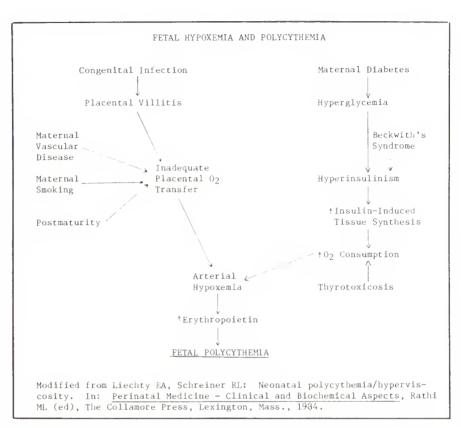


FIGURE 2

of cord clamping.¹² Most investigators report a 1-5% incidence in a general newborn population.¹

Hemoglobin and hematocrit concentrations are often significantly different in simultaneously obtained venous and capillary specimens with the latter as much as 12% higher, although considerable variability exists as demonstrated in *Figure 3.*13

The largest discrepancies are found in very premature infants (less than 30 weeks gestation) and in infants with compromised peripheral perfusion. This difference between "peripheral" and "central" hematocrits persists for as long as 10 weeks in premature infants and six weeks in term infants.

Ramamurthy and Brans have at-

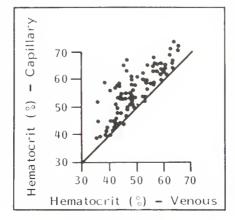


FIGURE 3: Scatter plot of simultaneously determined capillary and venipuncture hematocrit measurements. Note that while the capillary values are frequently greater, the variability precludes clinical usefulness. (Modified from Linderkamp O et al, Eur J Pediatr 127:9, 1977. Reprinted with permission from Liechty EA, Schreiner RL: Neonatal polycythemia/hyperviscosity. In: Perinatal Medicine—Clinical and Biochemical Aspects, Rathi ML (ed), The Collamore Press, Lexington, Mass., 1984.)

TABLE 2
Guidelines for Management of Polycythemic Infants

Peripheral venous Het <65%:	asymptomatic:	observe
	symptomatie:	consider hyperfibri- nogenemia, abnormal RBC deformability, acidosis
Peripheral venous Het 65-70%:	asymptomatic:	observe closely, fol- low glucose concen- tration and neurologic examination
	symptomatic:	partialexchangetrans- fusion
Peripheral venous Hct	partial exchange	

transfusion

tempted to determine the hematocrit at which hyperviscosity is usually present.10 They found that an umbilical renous hematocrit greater than 63% was strongly indicative of hyperviseosity. On the other hand, hematocrit determination on blood samples from heelsticks or peripheral venipunctures correlated less well with hyperviscosity. These data show the difficulties of accepting a hematoerit of 60-65% from a peripheral vein as a definition of hyperviscosity. However, an umbilical vein catheterization is an invasive procedure; therefore, most clinicians continue to use blood samples from a peripheral vein for the diagnosis of polycythemia.

> 70%:

During the first few hours of life there is a redistribution of plasma water which is associated with a decreased total blood volume and plasma volume. Red cell volume remains unchanged, resulting in an increase in the hematocrit and hemoglobin concentrations. The hemoglobin and hematocrit reach their peak concentration between one and 24 hours of age. (4.10)

Treatment

There is general agreement that symptomatic infants with polycy-

themia (hematocrit from a peripheral venous blood sample greater than 63-65%) should be treated by lowering the hematocrit to a more desirable range. This is accomplished by placing an umbilical venous catheter, an umbilical arterial catheter or peripheral artery and vein catheters and exchanging 10-15 ml aliquots of blood for either fresh frozen plasma or plasma equivalent (5% protein solution). If an umbilical venous catheter is used it should be above the diaphragm or in the umbilical vein (not in the portal vein or a branch thereof). The volume of fluid required to reduce the hematocrit may be calculated from the formula:

observed Hct-desired Hct observed Hct wt (kg) × 100 ml/kg*

*(volume of blood per kg of infant weight)

In contrast to the treatment of symptomatic infants with polycythemia, there is substantial controversy concerning the value of partial exchange transfusion in asymptomatic infants and in infants with mild nonspecific signs (e.g., mild lethargy or irritability). Several, but not all, investigators have noted a higher in-

cidence of prolonged neurologic dysfunction in asymptomatic polycythemic infants compared to controls. There is a paucity of data to help the physician determine whether or not partial exchange transfusion will prevent or modify this neurologic dysfunction. Therefore, the treatment of asymptomatic infants is speculative at this time. The clinician must establish his own guidelines for deciding when a partial exchange transfusion will be performed in the asymptomatic baby.

At our institution (Table 2) all infants have a hematocrit obtained on a capillary (heelstick) blood sample at one to four hours of age. Infants with a capillary hematocrit of greater than 65-70% have a hematocrit obtained on a peripheral venous blood sample. If the hematocrit on a peripheral venous blood sample is between 65 and 70% and the infant is asymptomatic, a glucose determination is obtained and the patient's clinical status is closely monitored. Other diagnostic tests to determine the etiology of the polycythemia are usually not indieated unless the history or physical examination is suggestive of a specific disorder (Table 1). If hypoglycemia is present or clinical signs of polycythemia/hyperviscosity are apparent, a partial exchange transfusion is performed. If the hematocrit from a peripheral venous blood sample is greater than 70%, a partial exchange transfusion is usually performed even in asymptomatic infants.

Summary

The significance of polycythemia and hyperviscosity in the neonate is still poorly understood. Considerable controversy regarding the diagnosis and treatment of neonatal polycythemia remains. Each physician must develop his/her own set of guidelines for the management of such infants.

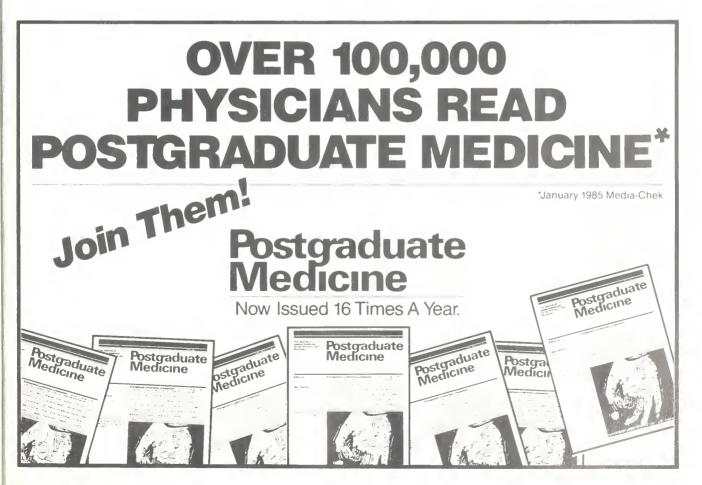
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Parental Stress during and after Pediatric ICU Hospitalization



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TUDIES OF STRESS and its alleviation in the intensive care environment most commonly relate sources and effects of stress to the patient and to intensive care unit (ICU) nursing and physician staff. Less is known about the psychological and emotional distress experienced by families of patients hospitalized in an ICU. For this reason and because of the time-consuming demands of providing sophisticated critical care, a thorough, organized, and timely understanding of and approach to stresses experienced by family members may be inadequate.

As pediatric admissions to the ICU are frequently unexpected, the illness severe, the outcome often uncertain, and the environment foreign, a high level of parental and family stress is common. Sources of parental stress must be identified and dealt with if optimum family support and ICU stafffamily relationships are to be realized.

In a "needs study" rather than research protocol format, we interviewed 20 parents of critically ill children over a 12-month period to determine sources of stress during and following their child's hospitalization in the Methodist Hospital Pediatric Intensive Care Unit (PICU). The children of parents interviewed were expected to be patients in the PICU for at least four to five days.

Parents were interviewed three

times: within three days of PICU admission; following transfer to general pediatric ward; and within one to three months after discharge or death (one of twenty patients). Interval questionnaires were designed to determine sources of stress and coping mechanisms at each stage of illness. Family history and recent life changes (e.g., job status, marital status, birth or death of family member) were evaluated to better understand parental reaction and coping mechanisms. Patient age was two months to 18 years; 14 were male and six were female. Diagnoses included: meningitis (3); multiple trauma (5); isolated head injury (5); severe burn (1); cardiopulmonary arrest (2); subarachnoid hemorrhage (1); Reye's syndrome (1); Vitamin K deficiency with acute hemorrhage (1); and multiple sclerosis (1).

Interview I

As expected, the medical condition of the child was the parent's primary concern during the first few days of PICU hospitalization. Uncertain prognosis and initial lack of information, or ability to assimilate information obtained, about the child's condition and its severity were described by most parents as the greatest initial stressor. Feelings of helplessness and overwhelming shock and disbelief were reported during this initial period.

Parents who saw their child in the outlying emergency room or Methodist Hospital Emergency Room, or who were present at the scene of an accident, generally were less emotionally traumatized by the patient's physical appearance at the parents' first PICU visit. Although most par-

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ents indicated that the PICU staff had described the child's appearance prior to their first visit, a physiologic reaction such as nausea, faintness, heart palpitations, or general tremor was common. One mother commented that "my legs just turned to jelly."

During the first 12 to 24 hours after admission, complaints included loss of appetite, irritability, muscle weakness, headaches, and abdominal pain. Feelings of general numbness, disorientation, and sleeplessness were also commonly experienced. Parents were generally so concerned for their child that they paid little attention to the significance of these reactions. They were consistently reassured to discover that these reactions were not uncommon. During these early hours, parents also experienced a sense of relief in knowing that their child had reached the definitive critical care environment.

The atmosphere of the PICU, including noise, lack of privacy, tension level, and unfamiliar equipment, was inevitably a source of stress. This atmosphere in some ways became a source of comfort rather than a threat to parents as their confidence and trust grew in the care provided by the staff. Many parents learned to read the various monitors, and reported feeling greater control and confidence as their participation in and understanding of the patient's care increased.

The patient's physical appearance, lack of opportunity to communicate normally with the child, and unfamiliarity with medical procedures led to feelings of alienation and displacement as the primary caregiver for their child. The close relationship that developed with the nursing staff was a source of comfort for most parents but occasionally aroused feelings of ambivalence. One mother stated that "it seemed like her nurse was more of a mother to her than I could be at that time." This mother's statement seemed to adequately summarize the feelings of helplessness, dependency, displacement, and relief that most parents expressed during the initial interview.

Guilt or self-blame, parental feelings of responsibility for the child's illness or accident, was a common response to PICU hospitalization. Some parents felt that they would be blamed by other family members and/ or the medical staff. Parental guilt was not always related to the immediate illness or accident. A mother whose 13-year-old daughter sustained a head injury from a bicycle accident stated that she always feared something bad would happen to her daughter because the mother had considered an abortion early in her pregnancy. Parents of another head injury victim were reminded by family members that this child was a result of an unplanned pregnancy and implied that this was God's way of punishing them.

Parents reported concern not only for their child but for other family members such as grandparents and siblings. What to tell other family members, how to tell them, whether other family members should be allowed to visit, and how they would cope with the news of the child's illness were concerns shared by most parents. Parents with other children at home frequently questioned what psychological effect this experience would have on the development of these siblings.

Frustration in trying to meet the needs of many family members was expressed, as was the parents' feeling of responsibility for keeping the family unit intact and functional even with a critically ill child in the PICU. All parents reported a feeling of overwhelming fatigue which they held responsible for their irritability and frustration with other family members and occasionally with the PICU staff.

Parents felt no hestitation in asking questions regarding their child's care although most parents felt more comfortable approaching the pa-

tient's nurse than the patient's physician. Some parents felt that the nurse was more optimistic regarding the patient's condition, some perceived the physicians too busy to interrupt, and others stated that the nurses were simply more available. All parents expressed a need to have daily contact with the physician even if the patient's condition was unchanged.

Some parents coped more effectively by asking many questions and gathering facts while others did well with little information and preferred not to be kept informed of test results and other details. An occasional parent felt a physician was avoiding them because he had bad news. One parent feared that the staff considered her an unconcerned mother because she did not visit often. Another parent felt that physicians were not spending enough time with her because her child was not as seriously ill as others.

Interview II

Parents with children in the PICU beyond one week expressed a feeling of strong dependency, security, and trust in the care provided by the PICU staff. Their gratitude and respect for the staff was apparent in their enthusiastic response to participation in the interview process. All parents were anxious to make a contribution to the staff in any way possible, and felt a strong desire to help other parents of critically ill children by sharing their own experience.

Because of these feelings of dependency and trust, the patient's transfer from the PICU to the general pediatric ward often caused anxiety and fear. One parent reported that she was afraid to leave her son's bedside on the regular nursing ward where no one seemed to appreciate how critically ill he had been or knew what the patient and family had been through. She missed the support from the PICU staff whom she had grown to know, and missed the empathy

from other parents whose children were critically ill at the same time. Parents were Irustrated by their perception of a decreased frequency of contact with the patient's physician once transfer occurred.

A new trust and security in nurses and physicians had to be established following transfer. We learned that support was critical at this time of increased parental responsibility for ongoing care of the child. Even more fatigue was experienced because parents felt more of a responsibility to participate in the patient's care. Some parents lelt insecure about nursing procedures that varied from those in the PICU. Parents often returned to the PICU parents' lounge to seek consolation from and offer consolation to parents who could most relate to their PICU experience. Some parents were annoyed by parents of their child's roommate who sometimes seemed inconsiderate of their feelings and of their child's life-threatening experience

Support from extended family members and friends occasionally dissipated once the patient was transferred from the PICU. Many parents felt very much alone at this time. One mother was outraged when a bridge partner called to inquire why she could not attend the bridge game that week as "her son wasn't that ill anymore." Parents invariably appreciated visits from the PICU physician and nursing staff after the patient was transferred.

Interview III

A major source of stress during the months following discharge was of a financial nature. Concern about where to go for help with the hospital bill, difficulty in communication with insurance and third-party payers, and surprise with physician fees was frequently expressed. Bills received for meals, transportation, and lodging during the child's hospitalization added to the financial difficulties. Disputes between insurance companies,

and the parental responsibility for providing insurance companies and financial agencies with adequate records and information was also mentioned as an ongoing problem for parents during the months following discharge. Several parents faced long and involved legal action related to the patient's illness or accident. Financial and legal issues were a lower priority at the time of hospitalization, but became a persistent source of anxiety long after the patient returned home.

Parents often expressed concern about behavioral changes and difficulties with the patient and/or siblings during the months after discharge. Some parents expressed guilt about not having spent more time with the patient's siblings during the course of hospitalization, especially when problems arose at home. Generally, parents stated that they had expected these difficulties as the family readjusted to normal functioning, and they were able to work through these problems without professional assistance.

Parents of head injured patients expressed the greatest number of concerns following discharge. Embarrassment and confusion resulted from the patient's erratic behavior, emotional dependency and liability, etc. Disappointment over the length of the recovery period and some fear of the future were expressed. Obviously, these families especially require ongoing support (family physician, pediatrician, Head Injury Foundation of Indiana, Inc.*) as they struggle with the practical and emotional aspects of caring for the headinjured patient.

All parents stated that they continued to relive the details of the illness, accident, and PICU hospitalization, especially at night. Some parents found it difficult to find supportive and understanding listeners during

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the months following discharge. Many corresponded in writing to the PICU nursing staff and to parents of PICU patients hospitalized with their child. It seemed that several months after hospitalization the full impact of the experience, whether positive or negative in outcome, was realized by the family.

The mother of a child who made a full recovery from a head injury was hospitalized for psychiatric treatment during the months following the patient's discharge. This mother had a history of depression, was experiencing marital difficulties, and faced an enormous unpaid medical bill. This is an example of how internal personal stresses affect parental ability to cope with the hospitalization of a critically ill child.

Internal sources of stress, and support systems within the family that predate the crisis of ICU hospitalization must be assessed to determine the degree and kind of help parents and families will need.

Discussion

The need to identify sources of stress to families of children hospitalized in an ICU, and ways to alleviate this stress, are in many ways obvious from our interview results. Additional thoughts can be found in what little literature there is on this subject. Parents interviewed made the following suggestions:

First and foremost, communication channels between ICU staff and parents should remain open at all times.

Parents wished to see the patient in the emergency room setting whenever possible. They requested that they be kept intermittently informed as to the patient's condition during extended surgery or procedures that exclude the parents' presence.

Parents emphasized the need to express their feelings to others who could listen non-judgmentally and to be selective in choosing their own support systems. They suggested that a support system be established

whereby parents of former patients could offer support to parents of new patients. Many interviewees volunteered their availability by telephone, and a parent-to-parent telephone call network was established and was felt to provide a special comfort to parents of subsequent PICU patients.

A tour of the general pediatric ward was suggested as one way of lessening the stress of transfer, as was introduction to other parents of former PICU patients and to new nurses

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and resident physicians.

Finally, parents suggested that contact be made by the PICU staff with the family after the patient returns home. Parents felt a need at this time to express their appreciation to the staff and to share their perception of the experience. As one parent stated, "I know it sounds funny but in a way I wish all parents could go through an experience like this. I don't think most parents really know what a gift their child is until some-

thing like this happens."

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Management of Vasospasm from Ruptured Intracranial Aneurysms with Induced Hypertension

Critical Care Medicine

TERRY G. HORNER, M.D. Indianapolis

Cerebral Vasospasm
Is the Most Frequent
and Serious
Secondary Complication
Likely to Develop
in Survivors of a
Ruptured Intracranial
Aneurysm . . .

UBARACHNOID HEMORRHAGE secondary to rupture of an intracranial aneurysm is one of the most dramatic and potentially devastating events in medicine. The hallmark of the acute bleed is the instantaneous onset of a severe headache.3 Some patients die or suffer irreversible brain damage at the time of the bleed. Of the many secondary complications that are likely to develop in the survivors shortly after the initial hemorrhage, cerebral vasospasm is the most frequent and serious. This brief communication describes how vasospasm is managed by the Indianapolis Neurosurgical Group with the aid of our medical colleagues.

Pathology

Fresh blood and clot from a ruptured aneurysm surround the cerebral blood vessels of the Circle of Willis, which traverses the spinal fluid filled cisterns at the base of the brain. As the clot undergoes lysis, chemically unidentified breakdown products may cause narrowing of the lumen of the involved cerebral blood vessels, a process referred to as vasospasm. This narrowing leads to increased vascular resistance, which impedes cerebral blood flow and may lead to brain ischemia or infarction.

Signs and Symptoms

Vasospasm should be suspected clinically when there is a decrease in

the patient's level of consciousness and/or the appearance of a new focal neurological deficit. Early manifestations may be subtle, and experienced clinicians and nurses are required to detect them. Patients who are noted to have a large volume of fresh subarachnoid blood in their cisterns on a CT scan performed shortly after the hemorrhage are prone to develop subsequent symptoms of ischemia. Signs of vasospasm usually appear about five to seven days after aneurysm rupture and may persist for several days to two weeks. Subclinical vasospasm may exist and may make intracranial surgery more hazardous. Our policy is to repeat cerebral angiography prior to contemplated craniotomy. The presence of significant vasospasm on the angiogram will usually result in postponement of the operation.

Management

Numerous medications shown experimentally to affect contractility of arterial wall muscle have been advocated for the treatment of vasospasm, but to date none has been clinically useful in its reversal.⁵ In our own practice the greatest success in overcoming the ischemic manifestations of vasospasm has been with the use of induced hypertension by volume expansion and, when necessary, vasopressors. We have become bold in using artificial elevation of the blood pressure in the preoperative patient

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prior to microsurgical ligation of the aneurysm, realizing the theoretical risk of precipitating aneurysm rupture.

Our treatment protocol is as follows:

- 1. All patients with subarachnoid hemorrhage are admitted to an intensive care or acute neurological care unit where they remain under constant observation by trained neurological nurses. The ischemic complications of vasospasm may appear abruptly and must be recognized immediately if treatment is to be effective.
- 2. At the first suspicion of symptomatic vasospasm, the neurosurgical team and the critical care physicians are notified immediately. Plasmanate or other appropriate volume expanders are administered rapidly. If the blood pressure does not rise with volume expansion, dopamine or dobutamine is started so as to raise the systolic blood pressure to a level just high enough to reverse or reduce the patient's neurological deficit. Arbitrarily we have not raised the blood pressure artificially over 200 mmHg systolic. It is not unusual to see hemiplegia disappear with elevation of the blood pressure and reappear with a drop in pressure.
- 3. Blood gases and electrolytes are drawn immediately to rule out a concurrent metabolic disorder.
- 4. Arrangements are made for emergency cerebral angiography by the neuroradiologists via the transfemoral route. We feel that ideally the diagnosis of vasospasm should be confirmed radiographically because of the risks involved in its management with hypertension.
- 5. Continuous intraarterial blood pressure monitoring is mandatory. Invasive cardiopulmonary monitoring via a pulmonary artery catheter is

- also usually necessary to assure that the intravascular volume is well expanded. Intubation and ventilatory assistance should be instituted at the first sign of respiratory embarrassment.
- 6. Complications of the patient's underlying disease and of this aggressive cardiopulmonary therapy (such as pneumothorax, thromboembolism, congestive heart failure, pulmonary edema and sepsis) should be anticipated and treated early.
- 7. In some normotensive patients and in patients with serious medical problems it is often difficult to induce artificial hypertension. We rely upon the ingenuity of our intensive care specialists to accomplish this feat. Also, if more than several days of induced hypertension is required, tachyphylaxis becomes a serious problem and increased dosages of pressors may be needed or other pressors such as norepinephrine added.
- 8. The need for continued induced hypertension is reassessed daily so as to maintain the blood pressure just at the level necessary to overcome the patient's neurological deficit. When the patient has been successfully weaned from induced hypertension, repeat angiography is performed to reassess the vasospasm and assist in determining the appropriate timing for operation.

Results

In my series of more than 260 patients with ruptured intracranial aneurysms, 34% developed clinically evident vasospasm requiring treatment with induced hypertension. Of these, 72% improved, 17% remained unchanged, and 11% continued to deteriorate. The rate of rebleeding in the entire group of more than 260 patients was 14%, but the rate in

those patients treated with induced hypertension was only 5%. This series compares favorably with that of Kassells, *et al*,⁴ except that many of their patients had been treated with hypertension only after clip ligation of the aneurysm.

Conclusion

Induced hypertension appears effective in the management of preoperative patients who develop symptoms of cerebral vasospasm following rupture of a cerebral aneurysm. The risk of rebleeding does not appear to be increased by this treatment. Ideally a drug will become available that will prevent vasospasm following subarachnoid hemorrhage. Some of the newer calcium channel blockers with cerebral specificity such as Nimodipine^{1,2} may be potentially useful for this purpose, but they are unavailable in the United States except for investigational use. We are presently participating in a multicenter study that is investigating one of these newer calcium channel blockers.

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Skin Diseases: Current Concepts, Therapy

1. ACNE

BRIAN POTTER, M.D. Michigan City

the incidence of skin disease more than doubles because of acne. Acne vulgaris is present to some degree in about 80% of adolescents of both sexes by the age of 17, the prevalence and severity increasing from about age 13 with the development of secondary sexual characteristics. Beginning at adolescence, endogenous androgens, comprising testosterone and its metabolites, occur in both sexes and exert a hypertrophic effect on sebaceous glands, leading to their enlargement and to the secretion of sebum.

Abnormal keratinization of the sebaceous follicle is one of the factors contributing to the etiology of acne. The earliest histopathologic change in acne is keratinization of the sebaceous gland duct and impaction of the funnel shaped follicle by dense, coherent, cornified cells. This produces the primary lesion of acne, namely the comedo, a plugged, dilated, sebaceous follicular infundibulum and orifice, visible on the surface as a blackhead or whitehead. The plug is formed from desquamated, cornified, squamous cells, the black pigment being melanin. Excessive keratinization below the follicular orifice during formation of the comedo leads to production of a compact, coherent, horny core (Figure 1).

After formation and maturation, the sebaceous cell moves from the germinative layer of the sebaceous gland to the duct. Here the cell disintegrates and releases sebum, which contains triglycerides. These become hydrolysed within the duct to fatty acids. Free fatty acids are comedogenic; injection or repeated application of them results in inflammation. and they appear to be chemotactic to neutrophils and macrophages. Rupture of the epithelial lining of the folliele enables its contents to escape into the surrounding dermis. This attracts large numbers of neutrophils, and stimulates the formation of foreign body granulomas, resulting in the papulopustules that are so evident in acne (Figure 2).

Another factor contributing to the pathogenesis of acne is colonization of the follicle by the anaerobe Propionibacterium acnes, formerly known as Corynebacterium parvum. This is not a pathogenic organism, and acne is not contagious, of course. However, acne may be regarded as a folliculitie associated primarily with proliferation of this micro-organism. Other organisms that can be isolated in culture from acne lesions include Staphylococcus epidermis (or albus), and Pi

tyrosporon orbiculare (or ovale), which also are nonpathogenic.

Propionibacterium acnes is part of the normal flora of the skin, but it is found in far greater numbers in the follicles of patients with acne. This organism accumulates in masses during the initial stages of comedo formation. It alters the process of follicular keratinization, either directly or by means of the bacterial products. These include lipases, antigenic proteins and extracellular enzymes, such as proteases, keratinases and hyaluronidase, the action of which can lead to follicular dissolution and clinical acne.

Patients with acne are more sensitive to Propionibacterium acnes than are normal persons. Injection of dead cell walls from this bacterium has the same effect as injection of the live organism, and even lipid-free cultural filtrates produce inflammatory lesions when injected into human skin. Inflammatory reactions may be potentiated by release of bacterial antigens during breakdown of follicular bacteria. These antigens are present within the follicles, comedones and inflammatory lesions. Acne subjects develop immediate type hypersensitivity to Propionibacterium acnes, with elevated serum antibody titers correlated with clinical severity. Among patients with cystic acne, some exhibit defective neutrophilic chemotaxis, while in others there is specific neutrophilic phagocytic dys-

Hormonal influence is another etiological factor. The development of acne is associated with urinary secretion of androgenic steroids and in-

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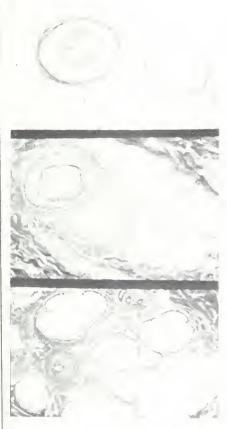


FIGURE 1: Sebaceous follicles impacted with keratinous plugs: just below skin surface (top), middle part of follicles (middle), and level of sebaceous gland (bottom).

crease in lipid levels on the skin surface. Conversely, the administration of estrogen results in suppression of the activity of sebaceous glands, independently of the gonads and pituitary and adrenal glands. Estrogens increase the rate of catabolism of sebaceous cells. Those estrogens that are active topically also exert systemic effects, through per cutaneous absorption.

The presence and recalcitrance of acne is not correlated directly with circulating levels of sex hormones, but presumably the sebaceous glands of the patient with acne are unusually sensitive to normal levels of androgen. Testosterone may be less com-

pletely metabolized in other areas of the skin than in those susceptible to acne, where metabolites are produced that have an enhanced effect on the follicles. These enlarged sebaceous glands are distributed chiefly on the face, upper chest and back, accounting for the characteristic localization of the lesions.

Treatment of Acne

Sulfur-containing lotions are still available, but they are no longer much employed, having become superseded by benzoyl peroxide. This compound has antiseptic properties, as do all peroxides, but has a longer duration of action than hydrogen peroxide. Benzoyl peroxide is supplied both as lotion and in the form of gels, in 2.5, 5 and 10% concentration. These may be applied once or twice daily.

Antibiotics are prescribed in acne to counteract the effects of the incriminated micro-organisms. The tet racyclines exert their antimicrobial action by the inhibition of bacterial protein synthesis, and inhibit the chemotactic responsiveness of human neutrophils."

Tetracycline, clindamycin and erythromycin are available for topical application as lotions. They have an effect similar to a low oral dose of the same antibiotic.

Several months of treatment are necessary to achieve clinical improvement. Blood chemistry and cell counts remain almost normal, except for minor variations, with a daily oral dose of 500 mg of tetracycline continued for a year or longer." Two grams can be given daily in divided doses if necessary for severe cases. Side effeets occur in 50% of such patients, but only about 10% have to discontinue treatment. Side effects include anogenital eandidiasis, gastrointestinal symptoms, liver dysfunction and occasionally Gram-negative superin fection.

Because the micro-organisms may develop resistance to antibiotics, in practice an antibiotic may fail to give



FIGURE 2: Follicular neck distended with pus (top); underlying sebaceous gland (bottom).

benefit after a time. When this hap pens, the maintenance dose can be increased to the maximum amount, or the treatment switched to another antibiotic.

The antibiotics ampicillin and tetracycline, much used in treatment of acne, decrease the effectiveness of oral contraceptives. In the case of ampicillin, this effect is due to microsomal enzyme induction. The mechanism of interaction between oral contraceptives and tetracycline has not been established. However, for patients taking oral contraceptives, erythromycin should be chosen as the antibiotic.

Retinoids related to vitamin A are now employed extensively in acne therapy. Retinoic acid (Retin-A) is applied topically for its keratolytic and comedolytic action." Even when high concentrations are applied there is no systemic toxicity from topical re-

tinoic acid. Much of the dose is degraded on the skin surface within one to two hours. Retinoic acid is a normal metabolite of, and is more rapidly metabolized than, vitamin A. It is totally degraded within 48 hours. A glucuronide conjugate is excreted primarily in the bile, with the side-chain intact.

With the oral retinoid isotretinoin (Accutane), sebaceous glands become markedly reduced in size, and the composition of skin surface lipids is considerably altered. Decrease of wax esters and squalene, and relative increase in cholesterol, indicate a lower contribution of sebum to surface lipid. The excretion rate of sebum can be decreased temporarily by as much as 90%, to only 10% of pretreatment levels, within three weeks.10 This is secondary to the reduction in size of sebaceous glands and inhibition of their differentiation. The count of Propionibacterium acnes is also reduced. This is, in turn, secondary to the reduced production of sebum, as the drug does not inhibit the bacterium in vitro.

These effects are dose-related. One tenth to 2 milligrams per kilogram of body weight can be given daily. The usual dose is 1 mg/kg/day, for three to five months. A higher dose is sometimes given for the first two or three weeks. The rate of production of sebum remains at about one-tenth of the pretreatment level until two weeks after discontinuance of the drug. Thereafter, it rises to about one third of the original level in eight weeks.

The mean time to peak plasma concentration of isotretinoin is approximately three hours. After an oral dose of 80 mg, the mean peak plasma concentration is 256 ng/ml. The drug is virtually entirely bound (99.9%) in the plasma, almost exclusively to albumin, by which it is transported to target cells. The elimination half-life is less than 24 hours. In some cases the blood concentration time profile is biphasic or triphasic, the secondary and tertiary peaks suggesting enter-ohepatic recycling of the drug.

The drug is concentrated in many tissues within 15 minutes, declining to undetectable levels within 24 hours, except for persistence as long as seven days in the liver, adrenal, ovary, ureter and lacrimal gland. 65-83% of a dose is excreted, almost equally in urine and feces. The major metabolite identified in blood and urine is 4-oxoisotretinoin, with some tretinoin and 4-oxo-tretinoin.

One of the principal mechanisms of action of these polar retinoids in high dosage is a detergent effect on cell membranes.11 Another is the disruption of lysosomes. Cellular retinoidbinding proteins, specific for retinol and retinoic acid, translocate to the cell nucleus and mediate other actions. The avidity of synthetic analogs for their receptors tends to parallel their biologic activity, which may be directly on the cell genome, to alter RNA synthesis. In the epithelium, only the basal cells are sufficiently undifferentiated to respond to the retinoids.

The side effects of isotretinoin include excessive dryness of skin and mucous membranes. More important is the definite teratogenicity of the drug, ¹² and a characteristic increase in serum levels of uric acid, triglycerides, cholesterol and phospholipids. Theoretically, this may predispose patients to arteriosclerosis, and could

precipitate a serious disorder secondary to hyperlipidemia, such as acute pancreatitis. The drug should therefore not be given to patients with a personal or family history of hyperlipidemia, nor to women of child-bearing age unless they abstain or take strict contraceptive precautions.

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BALANCED CALCIUM CHANNEL BLOCKADE!



Low incidence of side effects

CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

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Reduces angina attack frequency*

42% to 46% decrease reported in multicenter study.

Increases exercise tolerance*

In Bruce exercise test, control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes (P<.005).

CARDIZEM

(diltiazem HCl)

THE BALANCED
CALCIUM CHANNEL BLOCKER

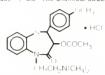
Please see full prescribing information on following page.

PROFESSIONAL USE INFORMATION



DESCRIPTION

CARDIZEM** (diltrazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltrazem hydrochloride is 1,5-Benzothiazepin-4(5H)jone,3-(acetyloxy)-5-12-(dimethylamino)ethyl-2,3-di-hydro-2-14-methoxyphenyl)-, monohydrochloride,(+)-cis- The chemical structure is



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 45D 98. Each tablet of CARDIZEM contains either 3D mg or 60 mg dilltazem hydrochloride for oral administration

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been

Anymid Due to coloniary artery Spasin CARDIZZIN has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.

Exertional Angina CARDIZEM has been shown to produce

increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads

In animal models, diltiazem interferes with the sfow inward I animal industry in the control of the configuration of the action potential tissues without changes in the configuration of the action potential Ditiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricu-lar conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses

In man, diltazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect, cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of dilitazem and beta-blockers. Resting heart rate is usually unchanged

or slightly reduced by diltiazem Intravenous diltiazem in doses of 2D mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20% In a study involving single oral doses of 300 mg of CARDIZEM In six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block Diltazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block in patients with sick sinus syndrome, diltazem significantly prolongs sinus cycle length (up to 50% in some cases)

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not considered showed showed sections.

usually produced abnormal protongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients:

Pharmacokinetics and Metabolism. Diltiazem is absorbed

from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bloavailability compared to intravenous dosingl of about 40% CARDUZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, aftered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin Single oral doses of 30 to 12D mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 2.5% to 50% as potient a coronary vasodilator as diltazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 mg/ml. There is a departure from dose-linearity when single doses above 60 mg are given, a 12D mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of dilitazem. impairment on excretion or metabolism of diltiazem

INDICATIONS AND USAGE

Angina Pectoris Due to Coronary Artery Spasm. CARDIZEM

is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks)

2 Chronic Stable Angina (Classic Effort Associated Angina). CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in

reducing angina frequency and increasing exercise tolerance. There are no controlled studies of the effectiveness of the concomitant use of dilutazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conductive. tion abnormalities

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 9D mm Hq systolic).

1 Cardiac Conduction. CARDIZEM profongs AV node refrac tory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow hear rates (particularly in patients with sick sinus syndrome) or second- or third-degree. AV block (six of 1243 patients for 0.48%). Concomitant use of dilitizer with beta-blockers or digitalis may result in additive. effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 6D mg of diltiazém.

Congestive Heart Failure. Although diltiazem has a negative notropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired verificular function is very limited. Caution should

be exercised when using the drug in such patients **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension

Acute Hepatic Injury. In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltrazem hydrochloride) is extensively metab General. CARDIZEM (United an injurior line) is extensively included by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity high doses of diltiazem were associated with hepatic damage special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver

which were reversible when the drug was discontinued in dogs, doses of 2D mg/kg were also associated with hepatic changes, however, these changes were reversible with continued dosing **Drug Interaction.** Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM (See WARNINGS

Controlled and uncontrolled domestic studies suggest that con-comitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 2D9

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed. in rats

Pregnancy. Category C Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 2D times the human dose or greater

There are no well-controlled studies in pregnant women, therefore, e_CARDIZEM in pregnant women only if the potential benefit

justifies the potential risk to the fetus

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation

Pediatric Use. Safety and effectiveness in children have not

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy

The following represent occurrences observed in clinical studies

which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their freguency of presentation, are edema (2.4%),

headache (2 1%), nausea (19%), dizziness (15%), rash (1 asthenia (12%), AV block (11%), in addition, the following e were reported infrequently (less than 1%) with the order of prestion corresponding to the relative frequency of occurrence

Cardiovasculai Flushing, arrhythmia, hypotension, brac

dia, palpitations, congestive heart fa syncone

Paresthesia, nervousness, somnole tremor, insomnia, hallucinations, and amn Nervous System Castrointestinal Constipation, dyspepsia, diarrhea, vom mild elevations of alkafine phosphatase, \$

SCPT, and LDH.
Pruritus, petechiae, urticaria, photosensi Dermatologic Polyuria, nocturia Other

The following additional experiences have been noted A patient with Prinzmetal's angina experiencing episodi vasospastic angina developed periods of transient asymptor asystole approximately five hours after receiving a single 6

asystice approximately live nours after receiving a single of dose of CARDIZEM. The following postmarketing events have been reported guently in patients receiving CARDIZEM erythema multiforms kopenia, and extreme elevations of alkaline phosphatase, § SCPT, LDH, and CPK. However, a definitive cause and effect bet these events and CARDIZEM therapy is yet to be established

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral dilitazem has been lin Single oral doses of 300 mg of CARDIZEM have been well tole by healthy volunteers. In the event of overdosage or exagge response, appropriate supportive measures should be employ addition to gastric lavage. The following measures may be considered.

Bradycardia Administer atropine (0 6D to 1 D mg). If is no response to vagal blockade, admin isoproterenol cautiously

Treat as for bradycardia above. Fixed degree AV block should be treated with High-Degree AV

diac pacing Administer inotropic agents (isoproter Cardiac Failure dopamine, or dobutamine) and diuretic Hypotension Vasopressors (eq. donamine or levarte bitartrate:

Actual treatment and dosage should depend on the severity: clinical situation and the judgment and experience of the tre physician.

Integral/L₅₀'s in mice and rats range from 415 to 740 m and from 56D to 81D mg/kg, respectively The intravenous L0, these species were 60 and 38 mg/kg, respectively The oral L1 dogs is considered to be in excess of 50 mg/kg, while lethality seen in monkeys at 36D mg/kg. The toxic dose in man is not k but blood levels in excess of 800 ng/ml have not been associated to the contraction of th with toxicity

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atheroscierotic (Exertional Angina Pectoris Due to Atheroscierotic Inary Artery Disease or Angina Pectoris at Rest Due to Inary Artery Spasm. Dosage must be adjusted to each palneeds. Starting with 3D mg four times daily, before meals a bettime, dosage should be increased gradually (given in didoses three or four times daily) at one- to two-day intervals optimum response is obtained Although individual patients respond to any dosage level, the average optimum dosager appears to be 18D to 240 mg/day There are no available data considered to the production of the p ing dosage reguirements in patients with impaired renal or he function if the drug must be used in such patients, titration should be used in such patients. carried out with particular caution

Concomitant Use With Other Antianginal Agents:

Sublingual NTG may be taken as required to abort a anginal attacks during CARDIZEM therapy Prophylactic Nitrate Therapy — CARDIZEM may be scoadiministered with short- and long-acting nitrates, but have been no controlled studies to evaluate the antial effectiveness of this combination.

3 Beta-blockers. (See WARNINCS and PRECAUTIONS.)

HOW SUPPLIED

Block

Cardizem 3D-mg tablets are supplied in bottles of 100 0088-1771-47) and in Unit Dose Identification Paks of 100 D088-1771-49. Each green tablet is engraved with MARIDN of side and 1771 engraved on the other CARDIZEM 60-mg stablets are supplied in bottles of 100 (NDC 0088-1772-47) and Dose Identification Paks of 10D (NDC 0088-1772-49), Each tablet is engraved with MARION on one side and 1772 on the

Another patient benefit product from



Cardiac Transplantation

Results of a Two-Year Experience at Methodist Hospital

HAROLD G. HALBROOK, M.D. LARRY H. STEVENS, M.D. DANIEL J. BECKMAN, M.D. Indianapolis

HERE HAS BEEN an active cardiac transplantation program at Methodist Hospital of Indiana since 1982. The efforts at Stanford University provided the impetus for the development of this program. Their relative standardization of the technique and improved survival results, with one- and two-year survival rates of 80% and 78% led to the conclusion that cardiac transplantation has moved from the realm of clinical investigation to therapeutic modality. The unit at Methodist Hospital is an attempt to deliver this service at the community level, in a non-university setting.

Materials and Methods

Between October 1982 and December 1984, 15 patients of 45 referrals underwent cardiac transplantation at Methodist Hospital. The criteria for recipient selection are presented in $Table\ 1$. Preoperative diagnoses included: ischemic, viral, familial and idiopathic cardiomyopathies. The criteria for selection of the donor organs are presented in $Table\ 2$. The donors ranged from 17 to 30 years of age. The causes of brain death were: gunshot wounds to the head (4), motor vehicle accidents (9), cerebrovascular

From the Graduate Medical Center, Methodist Hospital of Indiana, 1604 N. Capitol Ave., Indianapolis, Ind. 46202.

accident (1), and primary brain neoplasm (1).

Standard procurement techniques using systemic heparinization and a hypothermic, hyperkalemic cardioplegic solution were used. The Shumway-Lower method of orthotopic heart transplantation² was used in all cases.

Immunosupression for the first seven patients consisted of azathioprine, antithymocyte globulin, and corticosteroids. For the later patients in the series, cyclosporine, antithymocyte globulin and corticosteroids were used. For the first seven patients the diagnosis of rejection was based on the clinical status, serial EKG voltage measurements and endomyocardial biopsy. Since the introduction of cyclosporine, the routine use of endomyocardial biopsy has become the dominant technique for the detection of rejection.

The life table method of calculating actuarial survival was used.

Results

As of Dec. 1, 1984, 11 of the 15 patients were alive. Ninety-three per cent of the patients survived the perioperative period. The six-month survival rate was 84%. The one-year survival rate was 72% and the two-year survival rate was 52%. The Figure presents an actuarial analysis of the survival data. The one-year survivors spent 79.5% of their time out of the hospital. And, all of the survivors are NYHA class I or II.

There have been four deaths in the series to date. There were three deaths secondary to infection: 1) Staphylococcus capitis bacteremia and ARDS (11 days posttransplant); 2) Candida parapsilosis medistinitis (57 days posttransplant); and 3) Staphylococcus aureus pneumonia and acute

TABLE 1 Recipient Criteria

- 1. Less than 52 years of age; over 12 years of age
- 2. NYHA Class IV-Refractory to standard medical and surgical management
- 3. No insulin dependent diabetes mellitus
- 4. No evidence of malignancy
- 5. No recent pulmonary infarction
- 6. Pulmonary vascular resistance < 8 Woods units
- 7. No active infections
- 8. Psychologically stable

TABLE 2 Donor Criteria

- Meets established criteria for brain death
- 2. No history of cardiac disease
- 3. No evidence of systemic malignancy
- 4. No signs of systemic or intrathoracic infection
- Less than 30 years of age if male; less than 35 years of age if female
- 6. ABO compatible
- Negative lymphocyte crossmatch (recipient's serum with donor's cells)

renal failure (8 months posttran splant). The fourth mortality was eaused by a combination of acute and chronic rejection and occurred 12 months after surgery.

The incidence of rejection, infections, and complications were highest in the first three months posttransplant. There were 0.75 episodes of rejection per patient in the first three months. Bacterial, viral and fungal infections were encountered with an incidence of 1.13 per patient for the first three months after transplant. Other complications occurred at a rate of 1.40 per patient for the first three months postoperatively.

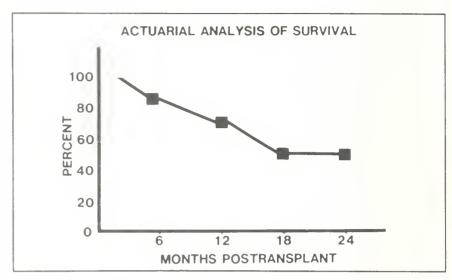
Analysis of the financial data reveals that the mean cost for the transplant admission was \$58,023; the range was \$25,310 to \$132,707.

Discussion

Immediately after the first human heart transplantation in December 1967 by Barnard at Groote Schur Hospital in South Africa, there was an explosive enthusiasm for the procedure. Approximately 150 cardiac transplantations were performed in the next two years. However, this early experience was largely unsuccessful. As the initial enthusiasm waned, only a few institutions continued active, clinical heart transplantation programs.

Stanford University has been one of the leading centers in the continued effort to improve results with cardiac transplantation, from the description of the technique of orthotopic heart transplantation in 1960 by Lower and Shumway² to the introduction of cyclosporine into cardiac transplantation by clinical trial in 1980. These efforts led to a 19% improvement in the one-year survival when comparing the period from 1968 to 1973 (44%) to the period from 1974 to 1981 (63%). With the advent of eyclosporine the one-year survival rate has increased to 80%.

The improved results at Stanford provided the impetus for the devel-



opment of the program at Methodist. This program is an attempt to apply cardiac transplantation as a therapeutic modality at the community level, thus making the procedure more readily available to the population. This also is less disruptive to the patient and his family, as no relocation is required and the waiting period between acceptance into the program and the operation may be spent at home. These goals have been accomplished with a one-year survival rate of 72%. This compares to one-year survival rates at other insti-

tutions ranging from 42% to >80%.7-11

With the standardization of techniques and improved familiarity with the expected course of the patients has come a reduction in the cost of cardiac transplantation. The hospital costs for the transplantation admission in the literature have decreased from over \$100,000 to an average of \$67,000.12 The cost for the transplant admission at Methodist averaged \$58,023. We conclude that cardiac transplantation can be carried out at a community hospital with acceptable survival rates and costs.

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SORBITRAT (ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows

INDICATIONS AND USAGE. A PRELITRATE lead enhanced in trate or in site of far the treatment and presented in dualities per hard. All desirgle forms of usoscolable declaratemay be used projettival for ally for her reuse frequency and diseventy of an ginut attacks and can be expected to

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dministered to air ursing woman.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been in tablished.

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotensium are distanced in sinicial trais at various drives, the following have been observed. Headache is the most common (reported inicidence varies widely apparently being disse related with an average incurrence of about 25% adverse reaction and may be severe in disease tested. C daneous vasoidation with flushing may occur. Pansient episodes of discrimensiand weak riess, as well as other signs of cerebral schemia associated with postural hypotensium, in maying associated with postural hypotensium, associated with postural hypotensium and sushing weakness restless riess pallor perspiration and collapse may occur even with the usual therapeutic dose. Drug such an dour extensive serious descriptions and collapse may occur even with the usual therapeutic dose. Drug such an dour extensive serious descriptions and collapse may occur even with the usual therapeutic dose. Drug such an dour extensive serious descriptions and collapse may occur even with the usual therapeutic dose. Drug such an dour extensive serious descriptions of methemoglobinema are rare at conventional doses of organic rutrate. The formation of methemoglobin is dose related and in the case of genetic abrormalities of hemioglobin that favor methemoglobin formation even conventional disease for subtingual SoRBITRATE in 15 foto 5 mg for chewable tablets, 5 mg, for oral (swallowed) tablets, 5 to 20 mg and for controlled release forms, 40 mg. SORBITRATE in 15 foto 5 mg for chewable tablets, 5 mg, for oral (swallowed) some approach and the conventional dose or side effects limit the dose in a subtingual SoRBITRATE in 15 foto 5 mg for chewable tablets, 5 mg, for oral (swallowed) salvets of the description and collapse for subtingual SoRBITRATE in 15 foto 5 mg for chewable tablets, 5 mg, for oral (swallowed) salvets of the description of the formation of the convention of the convention of th

SORBITIARTE should be intrafed upward until angina is relieved or side effects limit the dose is ambulatory patients. The magnitude of the incremental dose increase should be guided by measurements of standing blood pressure. The initial dosage of subtingual or chewable-SORBITRATE for prophylactic therapy in angina per foris patients is generally 5 or 10 mg every 2 to 3 hours. Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms.

demor strating the effectiveness of chronic maintenance therapy with these dosage forms have nit been reported.

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled release. Joues of 40 to 80 mg given every 8 to 12 hours is igenerally recommended. The extent to which development of tolerance should modify the dosage program has not been defined. The oral controlled release forms of isosorbide dinitrate should not be interest. DosAGE PORMS AVAILABLE: Sublingual Tablets (2.5.5.10 mg). Chewable Tablets (5.10 mg). Ural Tablets (5.10 mg). Sustained Action Tablets (40 mg).



PUBLIC HEALTH

CONTINUED FROM PAGE 360

at an alternative collection site and then referred to a physician for further evaluation. Third, physicians may see persons who apparently are not in one of the risk groups but who, after donating blood, were found to have a positive antibody test.

Finally, physicians need to be informed about AIDS so they can provide information on this frightening and often misunderstood disease to hospital personnel and other health care workers, as well as to the general public in their communities.

In the near future, clinical review of AIDS is scheduled to appear in this journal providing more detailed information on the disease. In addition, all physicians should have received information on AIDS and the HTLV-III antibody test in a letter from the FDA, as well as in a special issue of the Indiana Epidemiology Report. Copies of either or both can be obtained by contacting the Chronic and Communicable Disease Control Division, Indiana State Board of Health - 317/633-8414.

Membership Roster

The 1985-86 edition of the Indiana State Medical Association Membership Roster is now available. The roster will be mailed automatically to all ISMA members.

Additional copies of the roster will be sold for \$15.00 each for a physician member and \$30.00 each for a non-member, payable in advance. Checks should be payable to the Indiana State Medical Association.

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Neurologic Signs and Symptoms Related to Over-the-Counter Diet Pills

SHIRLEY M. MUELLER, M.D.*
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Abstract

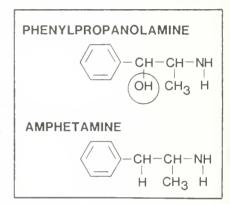
Five patients are described who had neurologic problems after taking over-the-counter diet preparations containing phenylpropanolamine and caffeine. All signs and symptoms, including blood pressure elevation, resolved when the diet preparations were stopped. This observation is important because over-the-counter pills are generally thought to be safe. Instead, they may be responsible for many otherwise unexplained neurologic signs and symptoms.

Many over-the-counter diet pills contain ingredients almost identical to "look-alikes" (Table 1). However, diet pills differ from "look-alike" pills in that most are time-released rather than immediate release. Therefore, medical complications that occur after "look-alike" pills would not necessarily occur after diet pills. In this communication we report five patients who ingested diet pills and suffered neurologic symptoms similar to the previously reported patients who ingested "look-alike" pills.

Patients (Table 2)

The acute symptoms ranged from irritability, restlessness and sleep-lessness (one Dexatrim) to a seizure (17 Dexatrim). The chronic symptoms were jitteriness, nausea and headaches (2-5 Dexatrim per day). Neurologic signs included dilated pupils, hyperactive reflexes and clonus. The blood pressure was elevated [x = 166 \pm 6 over 104 \pm 5 mmHg; (mean \pm SE)] in the four patients [x age 25 \pm

6 years (mean \pm SE)] who took more than one pill/capsule per day. All signs and symptoms, including blood pressure elevation, resolved when the diet preparations were stopped.



Discussion

Five patients developed the same neurologic signs and symptoms of "look-alike" pills after taking one or more over-the-counter diet preparations containing phenylpropanolamine in combination with caffeine. One patient also took diet "look-alike" pills. Recognition of medical complications associated with over-the-counter diet pills is important because their use is widespread and legitimate. Lack of recognition of such complications may lead to unnecessary morbidity or mortality.

In general, the symptoms were more severe when more diet pills/

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Supported by Grant PHS RO1 HL33126 01 from the National Institutes of Health. TABLE 1

"Look-alike" Pills/Capsules

Phenylpropanolamine 50mg Caffeine 200mg* (Ephedrine) (25mg)

Diet Pills/Capsules
Phenylpropanolamine
Caffeine 16

25-70mg 100-200mg

*As of Nov. 18, 1983, manufacturing of pills containing phenylpropanolamine and caffeine is illegal.

EUROPSYCHIATRIC SYMPTOMS have been reported in many people ingesting street "lookalike" pills containing phenylpropanolamine (PPA), caffeine and sometimes ephedrine. Phenylpropanolamine is similar in structure to amphetamine (Figure) and has been reported to have the same side effects.

TABLE 2				
Patient	Acute or Chronic Ingestion	History & Physical Exam	Blood Pressure	Family History of Hypertension
1	Either	37-year-old white woman complained of irritability, restlessness, and sleeplessness 1/2 hour after taking one Dexatrim for weight reduction.		Not applicable
2	Chronic	23-year-old white man complained of jitteriness, excessive sweating and nausea during the three weeks he was taking two Dexatrim (50 PPA/200 Caffeine) one day alternating with two Prolamine (37.5 PPA/140 Caffeine) the other for weigh reduction.	150/100 (100/70 previously)	Father with hypertension
3	Chronic	44-year-old white woman took up to five Dexatrim Extra Strength (75 PPA/200 Caffeine) every day for five months for weight reduction and developed severe headaches plus nausea.	180/120 (130/90 previously)	Negative
4	Acute	19-year-old white woman took 17 Dexatrim (type unspecified) in a suicide attempt. She vomited three hours later and had a generalized seizure. Dysarrhythmia was noted on EEG.	170/98	Not applicable
5	Acute	15-year-old white girl took three Dexatrim (type unspecified) and six "diet pills" which resem- bled "speckled pups" look-alike pills for weight reduction (?). Two hours later she became anxious and had sustained uncontrollable clonus and dilated pupils.	162/98	Not applicable

capsules were ingested. The 23-year-old man, however, had an elevated blood pressure when taking only two diet capsules per day. Both phenyl-propanolamine and caffeine cause central nervous system stimulation³ and increased blood pressure.^{3,5} Thus, in combination, the effect of one could be potentiated by the other.

Two of the patients with an elevated blood pressure (patients 2 and 3) may have had a predisposition to hypertension. One (patient 2) had a father with hypertension, and the other had a borderline hypertensive blood pressure of 130/90 mm Hg before starting the diet preparations. In animal studies, existing hypertension has been demonstrated to dramatically increase cerebrovascular

complications when PPA/caffeine was administered. Recognition of the association between diet preparation complications and a predisposition to an elevated blood pressure is important because hypertension is more common among overweight individuals. These people tend to take diet pills.

Four of the five patients reported took more than the recommended quantity of diet preparations per day. It is known that over-the-counter drugs are commonly taken in greater than the recommended dose since the general public assumes that they are safe. In addition, even when the recommended dose is taken, individual variability could lead to complications in one individual that would not

be found in another.

Effective Nov. 18, 1983, manufacturing of diet aids containing caffeine in combination with phenylpropanolamine was banned by the Food and Drug Administration until further notice." Nevertheless, the combination still remains on the store shelves until sold. In addition, ubiquitous caffeine can always be combined with phenylpropanolamine at the user's discretion.

In summary, neurologic complications of over-the-counter PPA/caffeine diet preparations are identical to those observed after "look-alike" pills. It is likely that PPA in combination with caffeine is responsible for many otherwise unexplained neurologic signs and symptoms.

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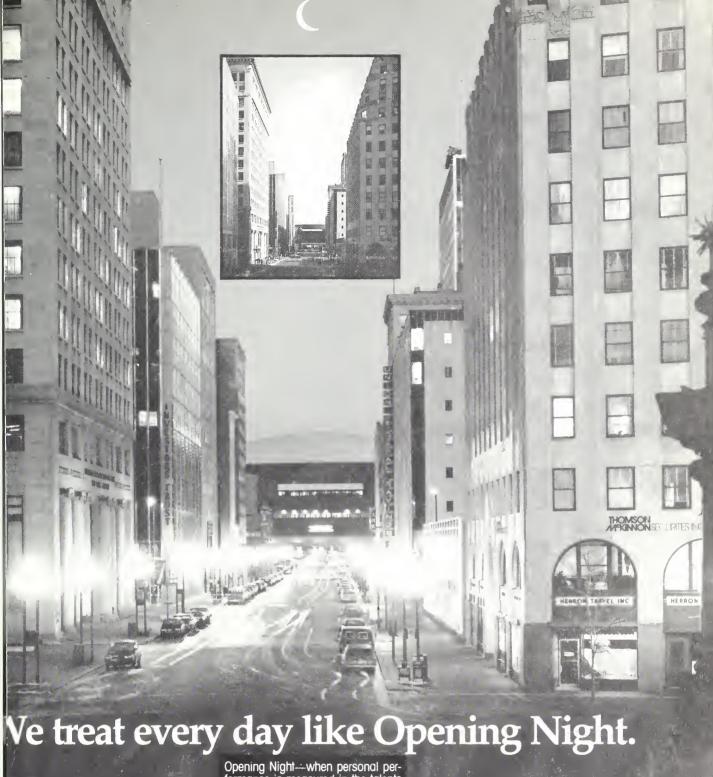
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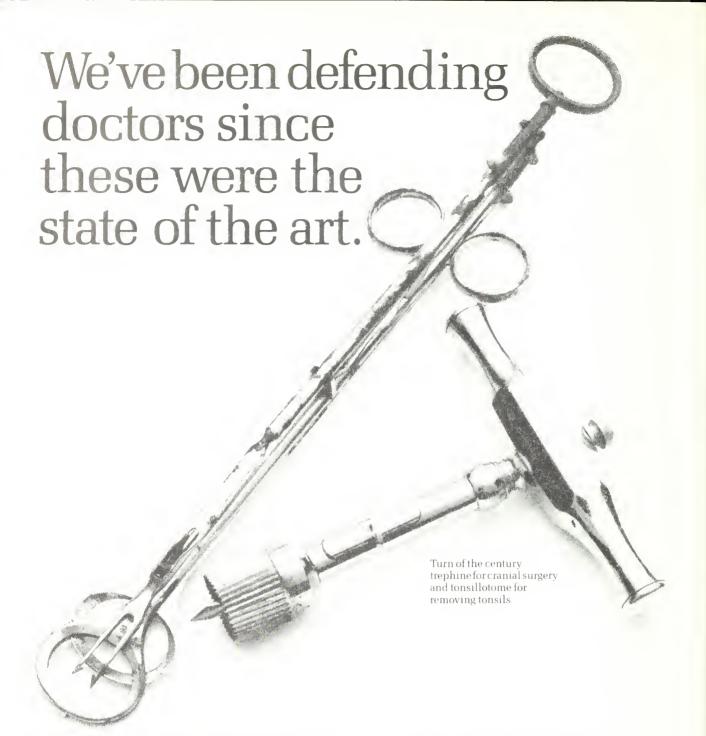
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Percutaneous Renal Stone Extraction: Experience with One Stage Procedures

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Abstract

Endourology is a rapidly growing field of interventional radiology. We describe the technique, results, and complications of percutaneous renal stone extractions from 57 kidneys of 55 patients over a period of eight months. Close cooperation between the radiologist and urologist is essential for success. Efforts to complete the extractions as one stage, one room procedures under general anesthesia have proven efficacious in our experience. We report an overall success rate of 95% and a ureteral stone success rate of 79%.

Key Words: Endourology, Nephrolithiasis, Nephrostomy Patient Selection

Almost any patient with calyceal, pelvic, or ureteral stones is a candidate for percutaneous extraction. A complete staghorn calculus is a contraindication only because of unavailable space for access to the upper tracts. Although more technically difficult, we have performed percutaneous stone extractions on patients with malrotated kidneys and horseshoe kidneys (Fig. 1). Ureteral stones located as low as the ureterovesical iunction have been successfully extracted. Patients with obstructive pyelonephritis underwent stone extraction following percutaneous nephrostomy catheter (10F) drainage and treatment of infection.

Technique

The procedures are done in the radiology department, preferably in an angiographic room with 105mm spot film capability and an L-U or C-arm configuration to allow imaging in multiple projections. The patient receives intravenous antibiotics for 24 hours prior to the procedure and is continued on oral antibiotics until the nephrostomy catheter is pulled out.

With the patient under general anesthesia, the urologist performs cystoscopy and places a two-lumen occlusion balloon catheter in the ureter just below the ureteropelvic junction. The patient is placed in the prone-oblique position with the side of interest up approximately 30°. The upper tracts are distended with contrast through the open lumen of the catheter after inflation of the ureteral balloon.

Many authors have reported their technique of obtaining access to the upper tracts.14 We attempt to stick a posterior lower pole infundibulum near the junction with its calyx with an 18 gauge styleted needle under fluoroscopy. Once position within the urinary system is documented by either aspiration of urine or injection of contrast, a wire is advanced into the pelvis (Fig. 2). The tract is dilated to 8F with angiographic dilators. If necessary, an angiographic catheter (H, or RC.) is used to direct the guidewire down the ureter to the level of the bladder. A long 8F non-hubbed catheter is advanced over the wire and a shorter 10F dilator is advanced over the long 8F catheter until its tip is directed down the ureter. The 8F catheter is removed and a second wire is advanced down the ureter through the 10F dilator. The 10F dilator is removed, leaving two wires with their tips near the bladder. One wire is used for tract dilatation; the second remains as a safety wire, assuring access to the urinary tract if the other wire is dislodged during manipula-

Tract Dilatation

Two methods for tract dilatation are currently available. One involves successive dilators and the other bal-

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Occlusion Balloon Catheter, 5F, 8.7 mm, 100 cm, Medi Tech.

Disposable Splenic Needle, 18 gauge, 15 cm, Cook Inc.

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Amplatz Renal Dilator Set - Cook Inc. Meadox Surgimed A/S, Oakland, NJ

Dow-Corning Corp., Midland, MI

^{*} Guardian Chemical, Smithtown, NY 11787





FIGURE 1-C

loon dilatation. We used the successive dilator system in 21 of 57 cases. Successive dilators are advanced under fluoroscopic control with a twisting motion over the long 8F catheter and guidewire. Olbert balloons were used in 34 of 55 cases (Fig. 3). We prefer balloon dilatation over successive dilators since it tends to shorten procedure time and produce less tract bleeding. The balloon catheter is backloaded with a 26F dilator and a 32F working sheath. Following balloon tract dilatation, the balloon catheter is advanced along the guidewire



FIGURE 1-A: The KUB film shows a 12x20mm stone in a lower pole calyx (arrow).

FIGURE 1-B: IVP of horseshoe kidney.

FIGURE 1-C: Post procedure KUB shows complete removal of the stone.

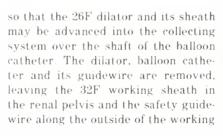


FIGURE 2: Drawing demonstrates a guidewire (solid arrow) being advanced through needle (open arrow) into the renal pelvis. Occlusion balloon catheter (double arrow) is identified at UPJ.

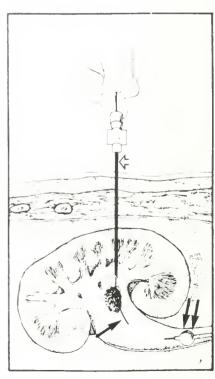




FIGURE 3-A: A 10mm Olbert Dilatation Balloon inflated with sharp indentation (arrow) at the level of the renal capsule is demonstrated. Occlusion balloon eatheter (1), safety wire (2), and wire (3) over which balloon catheter is advanced are identified.



FIGURE 3-B: After further dilatation, indentation of balloon is no longer seen.

sheath and extending down the ure-

Nephroscopy

After dilatation of the tract and placement of the working sheath, the urologist usually begins with the rigid nephroscope and forceps to remove blood clots from the collecting system. The working sheath maintains access as the instruments are withdrawn and also serves to tamponade the tract. Once the stone is visualized, and if it is small enough to pass through the working sheath intact, it can be removed by a variety of forceps or baskets which can be advanced through a port in the nephroscope.5.7 If the rigid scope is unable to locate the stone, a flexible scope is used. The flexible scope is capable of turning 160° upon itself.

Occasionally, fluoroscopy is used to guide the flexible scope into the stonecontaining calyx. If the stone is too large to be removed in one piece, an electrohydraulic or ultrasonic lithotripter can be used to fragment the stone. The electrohydraulic lithotripter was used in 18 and the ultrasonic lithotripter in six of our 57 cases. The electrohydraulic probe can be advanced through a port in the flexible nephroscope to reach calyceal stones not accessible to the rigid ultrasonic probe (Fig. 4). After fragmentation, forceps are used to extract the fragments. Details of the different kinds of lithotripters have been previously reported. 819

Post Procedure

Following removal of the stone, a large catheter, usually a 28-32 F Malecot*, is left in the tract with its tip in the renal pelvis. Besides providing drainage, the catheter also tamponades the tract to prevent excessive bleeding. If residual fragments of struvite stones are still present after the procedure, the patient can be treated with renacidin* irrigation through the nephrostomy tube. A nephrostogram is typically performed approximately seven days after the procedure to document any residual

nonopaque stones or extravasation. If normal, the drainage catheter is pulled. Tracts typically close rapidly with very little drainage after removal of the catheter.

Results

Percutaneous stone extractions were attempted on 57 kidneys of 55 patients. Forty-four stones were calcium oxalate, 11 stones struvite, one cysteine, and one uric acid. Stones were single in 63% and multiple in 37% of cases. If one considers all single stones and only the largest stone in cases of multiple stones, 37% were less than 10mm, 40% were 10-20mm, and 23% were greater than 20mm in size.

The procedure was successful in 54 cases (*Table 1*). Two of the three unsuccessful cases were ureteral stones which at surgery were embedded in the wall of the ureter. The third unsuccessful case was an early case in which successful dilatation of a nephrostomy tract was obtained; however, the nephrostomy tube fell out



FIGURE 4-A



FIGURE 4-C

while the patient waited two days for the stone extraction. It was decided to take the patient to surgery instead of attempting percutaneous stone extraction again. With our experience today, we would have performed the stone extraction on the same day as tract dilatation. Considering only the results of ureteral stones (*Table 2*), we were successful in 79% (*Fig. 5*).



FIGURE 4-B

FIGURE 4-A: 28F dilator (1) and 34F sheath (2) being advanced over 8F catheter (3) and guidewire (4). The safety wire (5) and occlusion balloon catheter (6) are also seen.

FIGURE 4-B: Tip of electrohydraulic lithotripter probe (arrow) pressed against stone.

FIGURE 4-C: Forceps (arrow) grasping final fragment.

This success rate is much higher than the 33% success rate on stones of the upper ureter reported by Amplatz, *et al.*¹

Complications

Complications of the 57 procedures are summarized in *Table 3*. Complications occurred in 25% of cases; however, only one complication (water intoxication) was considered major. This patient had successful extraction of an upper pole calyceal stone.

Following the procedure the patient had severe electrolyte imbalance, hemolysis, and acute renal failure requiring dialysis. The patient did recover renal function within two weeks. Leakage of hypotonic fluids into the retroperitoneum through the fornex of the calyx was thought to be the cause of the water intoxication.

Other complications included hemorrhage from the nephrostomy tract which was noted in seven (12%) cases. four (7%) requiring transfusion. Two patients had a perforation of the renal pelvis and one patient a perforation of the ureter. Both perforations of the renal pelvis occurred because we failed to have the guidewire and 8F catheter well down the ureter before attempting to pass successive dilators over them. The perforation of the ureter was secondary to a tear caused at the time of successful extraction of an upper ureteral stone with a basket. All three perforations healed spontaneously with external drainage of the collecting system. All complications lengthened hospital stay, but no patient was permanently impaired.

Discussion

Percutaneous renal stone extraction is a procedure that requires mastering of specific skills. A learning curve applies and approximately a dozen cases are needed to become comfortable with the technique. This learning curve is shortened by a cooperative effort between the radiologist and urologist. The physician team should therefore start with easier stone cases which include pelvic stones under 15 mm in size and lower pole calveeal stones. Large, multiple, upper pole calyceal, or ureteral stones should only be attempted after techniques have been mastered. Any stone, with the exception of a complete staghorn, becomes feasible once experience is gained.

Several institutions perform stone extractions as a two stage procedure.^{2,4,5} The first stage involves

TABLE 1 Results # PATIENTS SUCCESS n = 57PERCENT OF TOTAL COMPLETE 46 81% CLINICALLY Q 14% INSIGNIFICANT RESID **UAL FRAGMENTS*** UNSUCCESSFUL *Clinically insignificant residual fragments implies small fragments remained

> TABLE 2 Results on Ureteral Stones

which were thought very unlikely to cause the patient symptoms.

URETERAL STONES	# STONES n = 14	SUCCESS	PERCENT SUCCESS
ALL URETERAL STONES	14	11/14	79%
UPPER URETERAL STONES	5	5/5	100%
MIDDLE URETERAL STONES	5	3/5	60%
LOWER URETERAL STONES	4	3/4	75%

TABLE 3
Complications

COMPLICATION	# PATIENTS n = 57	PERCENT OF TOTAL
NONE	43	75%
HEMORRHAGE - TRANSFUSION	4	7 %
HEMORRHAGE - NO TRANSFUSION	3	5%
PERFORATION OF URINARY TRACT*	3	5%
PYELONEPHRITIS	3	5%
WATER INTOXICATION	1	< 20/o
URINOMA**	1	< 2%
*Healed spontaneously with drainage eath **Resolved spontaneously	eter in place	

placement of a small nephrostomy tube within the renal pelvis, usually performed under local anesthesia in the radiology special procedures suite. The second stage is usually performed in the operating room the next day and includes nephrostomy tract dilatation and stone extraction under general anesthesia. With the goal of shortening hospital stay, we attempted to complete our stone extractions as one room, one stage procedures. Urinary tract access, tract dilatation, and stone extraction are performed at the same setting under general anesthesia in the radiology special procedures suite. One room,

one stage procedures were performed in 82% (47 of 57) of our cases. These procedures can be time consuming to both the radiologist and the radiographic room.

We have performed procedures under both local anesthesia with intravenous analgesia and light general anesthesia. In our hands, the latter has been preferable since many patients poorly tolerate the long stays on the fluoroscopy table in a fixed position under local anesthesia alone. There have been no complications related to general anesthesia. The use of the radiology specials suite in our institution was less expensive and



FIGURE 5-A: 45-year-old man with a 3x4mm stone in the lower ureter.



FIGURE 5-B: Basket engaging balloon of occlusion balloon catheter.



FIGURE 5-C: Basket engaging stone in the ureter. Stone was successfully removed.

procedure scheduling easier than the operating room. Spot film capability was also beneficial.

Conclusion

Percutaneous renal stone extraction has been very successful at our institution with excellent results in extracting both renal and ureteral stones. Close cooperation between the urologist and radiologist is instrumental in optimizing patient care. The procedures are time consuming and require patience and determination to complete. Unsuccessful percutaneous extraction in three of 57 cases did not impair subsequent successful

operative lithotomy. The availability of extracorporeal lithotripsy will probably reduce the application of the percutaneous technique in the future.¹⁰

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INDICATIONS AND USAGE: These preparations are indicated for the treatment of infections caused by susceptible strains of designated microorganisms as follows: Respiratory Tract Infections (e.g., tonsillitis, pharyngitis, and lobar pneumonia) due to *S. pneumoniae* (formerly *D. pneumoniae*) and group A beta-hemolytic streptococci [penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever, Velosef (Cephradine, Squibb) is generally effective in the eradication of streptococci from the nasopharynx, substantial data establishing the efficacy of Velosef in the subsequent prevention of rheumatic fever are not available at present]; Otitis Media due to group A beta-hemolytic streptococci, *H. influenzae*, staphylococci, and *S. pneumoniae*; Skin and Skin Structures Infections due to staphylococci and beta-hemolytic streptococci; Urinary Tract Infections, including prostatitis, due to *E. coli, P. mirabilis, Klebsiella* species, and enterococci (*S. faecalis*).

Note: Culture and susceptibility tests should be initiated prior to and dur-

CONTRAINDICATIONS: In patients with known hypersensitivity to the cephalosporin group of antibiotics.

WARNINGS: Use cephalosporin derivatives with great caution in penicillinsensitive patients since there is clinical and laboratory evidence of partial cross-allergenicity of the two groups of antibiotics; there are instances of reactions to both drug classes (including anaphylaxis after parenteral use). In persons who have demonstrated some form of alfergy, particularly to drugs, use antibiotics, including cephradine, cautiously and only when absolutely necessary.

Pseudomembranous colitis has been reported with the use of cephalosporins (and other broad spectrum antibiotics); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with antibiotic use. Treatment with broad spec-

trum antibiotics alters normal flora of the colon and may permit overgrowth of clostridia. Studies indicate a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis. Cholestyramine and colestipol resins have been shown to bind the toxin *in vitro*. Mild cases of colitis may respond to drug discontinuance alone. Manage moderate to severe cases with fluid, electrolyte and protein supplementation as indicated. Oral vancomycin is the treatment of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile* when the colitis is severe or is not relieved by drug discontinuance, consider other causes of colitis.

PRECAUTIONS: General: Follow patients carefully to detect any side effects or unusual manifestations of drug idiosyncrasy. If a hypersensitivity reaction occurs, discontinue the drug and treat the patient with the usual agents, e.g., pressor amines, antihistamines, or corticosteroids. Administer cephradine with caution in the presence of markedly impaired renal function. In patients with known or suspected renal impairment, make careful clinical observation and appropriate laboratory studies prior to and during therapy as cephradine accumulates in the serum and tissues. See package insert for information on treatment of patients with impaired renal function. Prescribe cephradine with caution in individuals with a history of gastrointestinal disease, particularly colitis. Prolonged use of antibiotics may promote the overgrowth of nonsusceptible organisms. Take appropriate measures should superinfection occur during therapy. Indicated surgical procedures should be performed in conjunction with antibiotic therapy.

Information for Patients: Caution diabetic patients that fafse results may occur with urine glucose tests (see PRECAUTIONS, Drug/Laboratory Test Interactions). Advise the patient to comply with the full course of therapy even if he begins to feel better and to take a missed dose as soon as possible. Tell the patient he may take this medication with food or milk since G.f. upset may be a factor in compliance with the dosage regimen. The patient should report current use of any medicines and should be cautioned not to take other medications unless the physician knows and approves of their use (see PRECAUTIONS, Drug Interactions).

Laboratory Tests: In patients with known or suspected renal impairment, it is advisable to monitor renal function.

Drug Interactions: When administered concurrently, the following drugs may interact with cephalosporins:

Other antibacterial agents — Bacteriostats may interfere with the bactericidal action of cephalosporins in acute infection; other agents, e.g., aminoglycosides, colistin, polymyxins, vancomycin, may increase the possibility of nephrotoxicity.

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Carcinogenesis, **Mutagenesis**: Long-term studies in animals have not been performed to evaluate carcinogenic potential or mutagenesis.

Pregnancy Category B: Reproduction studies have been performed in mice and rats at doses up to 4 times the maximum indicated human dose and have revealed no evidence of impaired fertility or harm to the fetus due to cephradine. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, use this drug during pregnancy only if clearly needed.

Nursing Mothers: Since cephradine is excreted in breast milk during lactation, exercise caution when administering cephradine to a nursing woman.

Pediatric Use: Adequate information is unavailable on the efficacy of b.i.d. regimens in children under nine months of age.

ADVERSE REACTIONS: Untoward reactions are limited essentially to G.I. disturbances and, on occasion, to hypersensitivity phenomena. The latter are more likely to occur in persons who have previously demonstrated hypersen-

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sitivity and those with a history of allergy, asthma, hay fever, or urticaria

The following adverse reactions have been reported following use of cephradine: G.I. — Symptoms of pseudomembranous colitis can appear during antibiotic therapy; nausea and vomiting have been reported rarely. Skin and Hypersensitivity Reactions — mild urticaria or skin rash, pruritus, joint pains. Hematologic — mild transient eosinophilia, leukopenia and neutropenia. Liver — transient mild rise of SGOT, SGPT, and total bilirubin with no evidence of hepatocellular damage. Renal — transitory rises in BUN have been observed in some patients treated with cephalosporins; their frequency increases in patients over 50 years old. In adults for whom serum creatinine determinations were performed, the rise in BUN was not accompanied by a rise in serum creatinine. Others — dizziness, tightness in the chest, and candidal vaginitis.

DOSAGE: Adults — For respiratory tract infections (other than lobar pneumonia) and skin and skin structure infections: 250 mg q. 6 h or 500 mg q. 12 h. For lobar pneumonia: 500 mg q. 6 h or 1 g q. 12 h. For uncomplicated urinary tract infections: 500 mg q. 12 h; for more serious UTI, including prostatitis, 500 mg q. 6 h or 1 g q. 12 h. Severe or chronic infections may require larger doses (up to 1 g q. 6 h). For dosage recommendations in patients with impaired renal function, consult package insert.

Children over 9 months of age — 25 to 50 mg/kg/day in equally divided doses q. 6 or 12 h. For otitis media due to *H. influenzae*: 75 to 100 mg/kg/day in equally divided doses q. 6 or 12 h but not to exceed 4 g/day. Dosage for children should not exceed dosage recommended for adults. There are no adequate data available on efficacy of b.i.d. regimens in children under 9 months of age.

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ETHICS AND MEDICINE

1. Introduction

PAUL E. SCHMIDT, M.D. STANLEY J. MULLIN, M.Div. Indianapolis

Recent Incredible

Advances in Science

Have Made the Study

of Medical Ethics

a Burgeoning Field . . .

THICS IS THAT BRANCH of philosophy in which the process of decision-making in human activity is analyzed. Morality and ethics are not the same but are intricately intertwined. Morals are a set of societal rules/principles that govern human activity. These rules/principles are a way of defining what a society or portion of society values most highly and how individuals can act in order to attain the highest good in society.

Traditionally, each profession has also had a code of conduct. The code outlines the ideal basis for decision-making and activity. The code is often called "ethics." In this regard, ethics actually refers to a kind of morality required of members of the profession. Often, however, there are also structures included that provide means of analyzing activity of the organization in terms of its rules and regulations.

Medical Ethics: History

Hippocrates is considered the father of Western medicine and to him is attributed the Hippocratic Oath. The oath is a list of do's and don'ts that directs the physician on how to act in a human sense toward the patient. In general, the oath declares that respect for the patient, and not harming the patient, are the highest values to be obtained by the physician.

In the early Christian era the code was adopted and infused with a new spirit of mission and service by Christian physicians. Their calling to serve, in both Jewish and Christian context, is expressed in the covenantal ethic. In this case, the physician/patient re-

lationship goes beyond the contractural boundaries of law-beyond the technical expertise required in diagnosis and treatment—and includes the promise of fidelity. Fidelity is a mutual bond of trust between patient and physician. Fidelity does not eliminate disease or suffering but promises faithful and competent care.

This code of conduct, or ethic, influenced all of Western medicine and, in fact, was recited by graduating medical classes well into the current era. In the early and middle decades of this century, having "medical ethics" implied little more than codes of conduct between physicians regarding fees and the business aspects of medical practice.

Then, when it was discovered that many physicians participated (in psuedo-scientific experiments) in the atrocities of the Hitler regime in Europe before and during World War II, a true renaissance in medical ethics began. The World Health Organization of the United Nations responded by suggesting a modern code of conduct which might be applicable to all physicians in all cultural traditions. This code reaffirms the current decision to show respect and do no harm.

Medical Ethics: Today

Whereas it was the human tragedy of war in the 1930s and 1940s that stimulated a serious reexamination of the need for a strict code of ethics in the care of the sick, more recently it has been the incredible advances in science that have made the study of medical ethics a burgeoning field.

Many medical schools now have an

Correspondence: Methodist Hospital of Indiana, 1604 N. Capitol Ave., Indianapolis, Ind. 46206.

ethicist on the faculty. Courses in medical ethics are becoming a regular part of the curriculum and ethicists are even debating how much of a feefor-service they should charge. In a recent keynote address, the president of the AMA affirmed the primary role of medical ethics in the profession. The federal government has taken a similar position by sponsoring a major study into the ethics of medical care in this country. The potential loss by the profession due to governmental regulations is the reduction of the physician/patient relationship to a secular contract for service. Many new books on medical ethics have been published, periodicals offered and meetings convened.

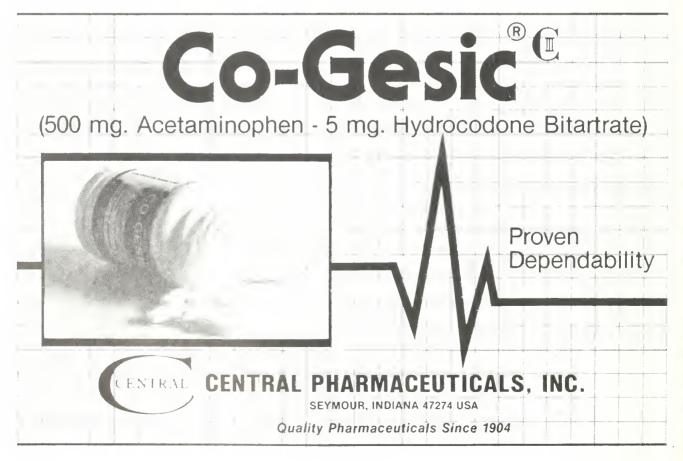
Since medical practice is still, at its core, a relationship between the patient and physician, there will always be a morality required. This review is the first in a series of reports on the current status of medical ethics. The next will discuss the structures, function and requirements of a hospital medical ethics committee.

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Notes from

The Royal College of Surgeons of England

AUSTIN L. GARDNER, M.D. Indianapolis

The Author Reviews

Several Scientific

Reports, Including

One That Deals with

a Severe Case of

Pretibial Myxoedema . . .

The Hunterian lecture in the November issue of the Annals of the Royal College of Surgeons of England by Frank Glassow was entitled "Inguinal Hernia Repair Using Local Anaesthesia." The technique was developed by Dr. E. E. Shouldice of Thornhill, Ontario, Canada and careful follow-up revealed a recurrence rate of around 1% in a series of 100,000 elective repairs.

The procedure is a modified Bassini, with strengthening of the internal ring and closure of the posterior wall using multiple layers of fine wire under local anesthesia, which allows evaluation of the repair during the operation. He cited Sir Cecil Wakeley, who stated, "Gentleness is the secret of success" and the operation "must restore structures in the inguinal canal as closely as possible to their normal conditions."

John Hunter, who had intimate knowledge of inguinal anatomy, would have been pleased with the results of this fine work.

A case of aortic ligation for iliac aneurysm performed by a Dr. Murray of the Cape of Good Hope in 1834 was presented. The first ligation had been performed by Astley Cooper in 1817. A left retroperitoneal approach under no anesthesia, by candlelight, in the patient's bed, reminds us of the great changes that have occurred in the last 150 years.

The patient complained during the operation of the other leg "becoming as benumbed and useless as his (right), and that we had done him a bad service in laming his good leg ... and lamented it bitterly." The patient died 23 hours after the operation.

Ligation and compression remained the treatment of aneurysm until Matas introduced endoaneurysmorrhaphy in 1888, and Goyanes performed a vein graft after resecting a popliteal aneurysm in 1906.

"Experimental Deep Venous Thrombogenesis by a Non-Invasive Method," by John D. Hamer and P.C. Malone of Birmingham, was an important contribution. Experimental studies in the past did not simulate deep vein thrombosis in the human. The authors noted that venous thrombosis could be prevented in the anesthetized dog by gentle passive motion. They felt that thrombus formation occurred in the valve pocket due to hypoxia and that deep vein thrombosis always started in the valve pocket if there were no preceding endothelial injuries.

"Relief of the Pain of Unresectable Carcinoma of the Pancreas by Chemical Splanchnicectomy During Laparotomy" was presented by A.M.N. Gardner and G. Solomon of Devon. 40 cc of 5% phenol in almond oil were



FIGURE 1-A

injected behind the aorta approached through the lesser sac with relief of pain in 81% of a series of 41 patients. The authors felt that this was safer than percutaneous injections.

Six cases of pretibial myxoedema were reviewed by G. Stewart of St. Thomas Hospital, London with the late J. B. Kinmouth and N. L. Browse. The authors found the condition in both hyperthyroid and hypothyroid state. This condition had never been investigated by lymphangiography, which had been developed by Professor Kinmouth, and the studies were essentially normal. It was felt that since myxoedema is used to describe



FIGURE 1-B

clinical features of hypothyroidism and edema implies an excess of watery fluid, the condition might better be called pretibial mucinous infiltration.

The accompanying figures, reproduced from the *Annals*, show two stages of a rapidly progressing, severe case of pretibial myxoedema. *Figure 1-A*, taken in 1978, shows diffuse thickening over the leg and foot; *Figure 1-B*, taken in 1981, shows the progression of the disease to an elephantiasis-like appearance—gross swelling, thickening and multiple nodules; *Figure 1-C*, also taken in 1981, provides a close-up view illustrating the nodularity and the depth of the crevices.



FIGURE 1-C

Many other interesting articles were published. A study in gynecological patients operated with and without masks by operating room doctors and nurses revealed a 60% incidence of infection in the unmasked group and no infection in the masked group. The Ethics Committee gave permission for the trial and evidently discontinued the study when the results disproved the implication of a study by N.W.M. Orr suggesting that masks were not necessary. The difference was explained by the fact that the unmasked doctors were chosen for their volubility and were encouraged to talk.



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Are Angioplasters Split?

Commentary

THOMAS E. ROSE, M.D. Evansville

T THE 34th ANNUAL American College of Cardiology sessions, March 1985, experienced centers presented data on multilesion/multivessel coronary angioplasty. There appeared to be a "split" among the experts as to how aggressively multivessel and multilesion coronary angioplasty should be approached. I believe from close inspection of the philosophies of the centers, review of their technical differences and approaches, this emerging controversy can be better understood.

Ever since Dr. Andreas Gruentzig dilated the first human coronary artery in Zurich in 1977, the procedure has been evolving into an accepted form of treatment. With guidance from Dr. Gruentzig, the NIH's Heart, Lung and Blood Institute set up a PTCA registry in 1979, which documented the results of primarily single vessel procedures until 1980. The data collected until September 1981 included a small number of multivessel cases, also. The Registry closed to complex angioplasty, including multivessel disease, in September 1981.

The predominant influence throughout the registry period for PTCA indications and technique was Dr. Gruentzig's group. By late 1981, Dr. Gruentzig had moved from Switzerland to Emory University in Atlanta, Georgia.

My personal experience with PTCA began with study with Dr. Gruentzig in Zurich in 1980. Deaconess Hospital (Evansville, Ind.) entered the NHLBI registry in 1980 as the 59th site. Dr. John Oak and I contributed to the initial single vessel data as well as the later multivessel data collected until closure of the Registry in September 1981.

Dr. Gruentzig's extensive contribution to this early angioplasty period can neither be underestimated nor over appreciated. He not only contributed generous amounts of time in training other cardiologists in PTCA procedures, but established the early techniques and guidelines. Significant among these guidelines were the use and measurement of pressure gradients across coronary lesions and advice not to recross dilated lesions. These two guidelines become important in understanding development of the current controversy.

In approximately 1981, new approaches to angioplasty began to appear, such as the concept of balloon catheters with moveable guidewires later to have the capability of "steerability." The guidewire concept allowed much greater ease in crossing tight stenoses and distal stensoses and greater safety in multilesion/multivessel cases. Despite these advantages, however, the new system made it difficult to measure pressure gradients as previously practiced. With this guidewire system, several centers began actively investigating multilesion/multivessel cases.

Probably the greatest advocate of

the guidewire system, Dr. Geoffrey Hartzler of Kansas City, has collected significant data in previously untested areas of angioplasty - including multivessel PTCA, multilesion PTCA, PTCA in AMI and patients who are not surgical candidates. Dr. Hartzler's success established the advantage of the guidewire system. The Gruentzig balloon system was modified to a guidewire system. The modification done to the Gruentzig system retained the ability for pressure measurement. The extra port necessary for pressure measurements made the balloon catheter stiffer and harder to navigate turns in the coronary tree. Its larger balloon diameter can make it more difficult to pass tight or distal coronary

Philosophical differences have also evolved. A significant difference is whether to recross coronary lesions once dilated. This is crucial when a dilated vessel abruptly recloses, but also applies to recrossing and redilating lesions that appear less than optimally opened during multivessel cases. Dr. Hartzler advocates recrossing dilated vessels that abruptly reclose and has a 90% success rate. This corresponds to my own experience.

It is my belief that the technical and philosophical differences of PTCA have led to different experiences at various centers. The apparent conflict of multivessel/multilesion PTCA indications can be attributed to these differing experiences.

The Controversy

At the American College of Cardiology meeting, Dr. Gruentzig's group

From Evansville Cardiology Associates, Inc., 350 W. Columbia St., Suite 400, Evansville, Ind. 47710.

presented data on multivessel PTCA and recommendation for its use.1 They excluded all patients with left main disease, poor LV function, one totally blocked coronary artery, long or eccentric lesions or patients with valvular heart disease or previous CABG or AMI. Despite these exclusions of "high risk" patients, only one lesion was dilated in 344 of 457 patients with multivessel disease. Two lesions were attempted in the remaining 113 patients. Primary success ranged 81-86%, death rate was low (0.4%), but acute closure rate was 6.5% in single dilations and 9.7% in multiply dilated patients. This led to an overall 5% emergency surgery rate. In the question-and-answer session, the presentor stated that many of the abrupt reclosures were not recrossed. but taken to emergency surgery. Based on their experience, they suggested great caution in applying PTCA to multilesion/multivessel cases.

Dr. Jerald Dorros of Milwaukee presented data² on over 600 cases with multivessel disease, dilating more than one lesion. He included patients with left main disease; over 40% had impaired LV function, approximately 20% had previous bypass, and a large number had AMI, COPD, chronic renal failure or were elderly. He reported an emergency surgery rate less than 2.5% and mortality less than 1%. Primary success was greater than 90%. He

found PTCA useful and highly successful in a much sicker patient population than Dr. Gruentzig's group reported.

Dr. Geoffrey Hartzler had presented data³ on the largest group of multivessel dilation—up to 10 lesions in one patient. His patients also included several with poor LV function, previous CABG and severe associated diseases. He reported a 90% primary success rate, with 1.7% emergency surgery and 1% mortality in over 1,200 multivessel patients.

My own experience with multivessel/multilesion angioplasty is similar to that of Dr. Dorros and Dr. Hartzler. Using similar technical and philosophical approaches, I have developed a 93% primary success rate in multivessel angioplasty.

Conclusions

Controversies in clinical medicine usually lead to better patient care. Many centers are reporting excellent results from PTCA applied to complex cases, including multilesion and multivessel cases as well as angioplasty in AMI, patients who have had previous CABG, and patients who are not surgical candidates due to associated medical diseases. There do exist, however, differences in the suggested indications from the various centers for multivessel/multilesion PTCA.

I believe as more data accumulate, angioplasty will be shown to be the correct alternative of therapy for an expanding number of patients with coronary disease. As more invasive cardiologists gain experience with multivessel PTCA, and more cases are added to the reports of complex PTCA, angioplasty will be indicated in 40-50% of patients currently treated with CABG.5 Hopefully, similar to the guidewire controversy of 1980-81, a predominance of data will clarify the indications for angioplasty in multivessel PTCA. Then we will see a "healing" of the current split among angioplasters.

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Life expectancy in this country has risen from 69.7 to 74.5 years since 1960 for all males and to nearly 79 years for women. Infant mortality has been reduced to a record low of 10.9 deaths in the first year of life for 1,000 live births. This represents a steep decline from the figures of 24.7 deaths per 1,000 live births in 1965, and 16.1 in 1975. Polio has almost been eliminated. The incidence of mumps has fallen from more than 150,000 cases, as recently as 1968, to just over 3,000 last year. Cases of measles have dropped from nearly 1/2 million in 1962 to 1,400.

Since 1970 deaths from heart disease are down 25% and from stroke 40%, due largely not only to major technical advances, including open heart surgery, pacemakers and new drugs, but also due to greater public awareness of proper exercise, diet and lifestyle.

Cancer is still a major threat, but patients are living longer after treatment, and many forms of cancer once fatal, are now curable.

Transplant surgery aids people who otherwise would face long hospitalization, serious disability, or death. One hundred Americans a year receive new hearts, 5,000 receive new kidneys. In 1983 alone 23,000 cornea transplants restored sight to people whose vision was severely impaired and 65,000 artificial hip replacements brought relief from disability and pain suffered by pa-



LAWRENCE E. ALLEN, M.D. President Indiana State Medical Assn.

tients with chronic hip disorders.

New diagnostic devices that give clear views of internal organs and systems have greatly enhanced the physician's ability to make rapid and more accurate diagnoses. They include computerized axial tomography, known as the CAT Scan, and more recently we have seen the development of magnetic resonance imaging, or MRI. These devices often eliminate the need for exploratory surgery or other invasive procedures.

Genetic engineering has produced human insulin for use in medicine and holds enormous promise for other future similar developments. Since 1960 other significant new developments have included the heart-lung machine, which has made open heart surgery possible, the surgical attachment of severed limbs, ultra-sound diagnostic devices, compact kidney dialysis machines for home and ambulatory use, multiphasic diagnostic screening, computer enhanced vital sign monitoring systems, prosthetic devices to replace blood vessels, heart valves, bones and joints, and thousands of others.

Approximately 90% of the drugs and medications that physicians can prescribe for the benefit of their patients today were unknown only 25 years ago.

We have, in fact, banked up a tremendous storehouse of medical technology, designed to let us live longer and more comfortably and enjoy an enhanced quality of life. Such new technology, however, will make medical care cost more in the future, but the alternative of letting people die sooner and live with pain and disability seems an alternative totally unacceptable to the American public and the physicians and hospitals that provide medical care. The choice, nonetheless, is with each of us, and the way in which we exercise that choice will determine the future of medicine.

If we are to look to a single determinant in guiding our health care choices and that single determinant is cost, we must be aware that, as John Ruskins wrote some 150 years ago, "There is nothing in the world that some men cannot make a little poorer and a little cheaper and those who look at price alone, are his legitimate prey."

Our present system has given us longer, healthier lives at a net savings to society. This is easily seen when we consider the improved treatment resulting in one million fewer people housed in mental hospital facilities with an estimated \$35 billion being saved each year. This is the result of pharmaceutical research and improved psychiatric techniques of management,

all of which lend themselves to the ambulatory care of the psychiatric patient. Another example of huge savings to our society through medical research and improved medical treatment, is the result that many thousands fewer people require therapy in Tuberculosis sanitariums, with at least \$20 billion being saved each year. Consider also the fact that through improved medical management of patients with coronary artery disease, even though the cost of such therapy may be significant, many thousands of coronary artery disease patients have recovered as a result of advanced diagnostic and therapeutic technology and methods of care. They have returned to work where their earnings within the first six months of being back on the job have more than paid for the cost of their medical care. Consider also the patients who are today cured from formerly fatal forms of cancer, who are now in our work force. generating income, paying taxes and contributing to the general welfare of our society. A health care system that brings about these kinds of results cannot be considered a liability to society. That is without question society's greatest asset.

If medical care deteriorates in this drive for economy that is now so prevalent, the most pressing question at the end of the next five years will be WHO IS TO BLAME and HOW DO WE RESTORE OUR PRIORITIES? If I were a public official, I would not want to be known or remembered as one of the architects who designed the DISMANTLING OF OUR HEALTH CARE SYSTEM.

Certainly U.S. medicine cannot do everything for everybody, but many obligations do lie ahead, and one such obligation has to do with the increasing population of older citizens and the fact that they consume and need more health care than any other segment of

our population. They, in fact, use 30% of all health care that is consumed by the American public. If we as a society are to morally respond to this future of increased health care need for our older citizens, the cost of health care making up the gross national product may very well increase from its present level of 10.5 to 11%. How Americans spend their money and what priorities they exercise in doing so is, in fact, their national privilege and is not inconsistent with the assessment that qualities such as good health and good education belong at the top of the priority list for our nation as well as the individual.

In satisfying our projected health care needs, we cannot, however, lose sight of the reality of our resources. How efficiently we learn to employ our medical technology and services will determine how likely future inevitable forms of health care rationing will affect costs versus benefits.

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EDITORIALS

Who's Biting the Bullet? It 'Hardly Seems Fair'

Letter to the Editor

I am an internist of 40-plus years of experience. I am in continuous solo practice in an average American town. I have a record of continuity of care for thousands of people, including many members of extended family groups, even into fourth and fifth generations of some families. I maintain excellent, chronologie, dictated, typed records which colleagues and visiting resident physicians tell me are without local equal. I am a concerned, prudent physician, I maintain a neat, clean, comfortable, non-fancy office. I have three fulltime energetic employees, and several part-time employees. I live in a nongrandiose country home. I drive a Dodge truck. I have always in the past enjoyed this type of practice.

At the request of the American Medical Association and the American Society of Internal Medicine, I voluntarily began a fee freeze on January 1, 1984. My fees at that time were in many instances significantly less than those of other internists in my town. Then came the Budget Deficit Reduction Act (Public Law 98-369) that required that I freeze my Medicare fees at the April-June 1984 level, which of course in my case meant the January 1984 level.

To be accurately sure that my Medicare patients' fees are those of June 1984, I have had to make major

business record keeping changes, including fractionating the type of service into all the various code numbers using a new "superbilt." This has required large amounts of time, the time of my secretary, the time of my bookkeeper, and the time of a hired outside expert in such record keeping systems. The new Medicare regulation has already caused my overhead to increase. Further, all the study time that I have had to spend on this fee freeze problem has taken away from my study of new scientific advances in the field of medicine. One of my academic colleagues tells me that the years 1984-1985 will be looked back upon as the "stupid years," because in that period of time doctors read less of their scientific journals and more of the economic tracts.

Also, quite obviously, the solo practitioner has been much more bothered by this regulation than the group practitioner because he has to figure out his own pathway through the red tape maze; whereas, the group man can follow the advice of a hired expert in bureaucratic maze running.

And, inasmuch as I have been in practice for over 40 years, I have a large fraction of Medicare patients in my practice. Freezing Medicare fees has effectively leveled by gross income, although in the same period of time, my expenses in equipment, services and salaries have continued to go up steeply.

My accountant now tells me that I

will have to consider either cutting salaries, or reducing the quality or time of my services to my patients. Both of these seem unacceptable to me.

So, I remain in an unhappy quandary as I write this fetter. So far my only token resistance to the economic bind has been to tell my receptionist to refuse the request of any prospective new patient over the age of 60 who calls in, asking to become a patient of mine. I worry some that this may mean that Medicare patients may find it difficult to locate doctors, just as Medicaid patients have sometimes in the past. Also, I have increased the fees of my non-Medicare patients this year more than I expected necessary in order to cover my rising overheard. This means that my non-Medicare patients are to some extent subsidizing my Medicare patients, but this hardly seems fair. -(The writer is a respected internist who has asked to remain anonymous.)

To Join or Not to Join Open Letter to Physicians

This is a time when delivery options for medical care are changing rapidly. With the advent of PPOs, HMOs and IPAs, an increasing choice is being offered to patients. Some of the plans which are proposed or are currently being offered may benefit the patient. Some of these plans, however, may be detrimental to the best interest of your patient.

Currently Slavery and Indentured Servitude is still illegal in the United States and this should apply also to physicians. Physicians are not required to serve in any of the PPOs, HMOs or IPAs if they do not wish to or if they believe that these plans will result in inferior care being delivered to their patients.

Currently, freedom of speech exists in the United States and this still applies to physicians. Physicians may voice their praise or opposition for any particular health care delivery system.

All of the health care delivery systems depend upon physician participation in the plans. If a physician feels that any particular PPO, HMO,

ISMA Constitutional Amendment

As required by Article X (Amendments) of the ISMA Constitution, Indiana Medicine announces the following Constitutional amendment:

Resolution 84-6 (Subject: Article III—ISMA Constitution—Component Societies) was introduced by the ISMA Commission on Constitution and Bylaws during the 1984 session of the House of Delegates. The resolution, which follows, was adopted. The boldface type indicates changes to the present language.

"Resolved, That Article III—Component Societies, be amended to read: Component societies are those county, district or other medical societies as specified in the bylaws contained within the state of Indiana, and who hold charters from this Association."

IPA, or other system may be detrimental to his patients, he may vote on this system by refusing to join or by resigning from that plan. If enough physicians feel the same way, the plan will fail for lack of physician participation.

If physicians feel strongly about any particular plan, they can vote for the plan by participating. The physician's participation or lack of participation will make or break any particular health care plan. Since new PPOs, HMOs, and IPAs are being formed continuously, a physician should not feel pressured to join all of them or any of them. Each physician may fear the loss of patients or reduction in income. Physicians must have the courage to stand by their convictions if they feel a particular plan is not beneficial. If the physician is proven wrong, he can then join that plan at a later date and restore his patient volume and income. Physicians should not feel pressured, however, to join any particular plan, even if a large organization is behind it.

These views are solely my own, and are made under Constitutional freedom of speech. This letter is not an effort to dictate policy or to institute

restraint of trade. This letter is being provided to open a forum for discussion on these topics.-Randolph W. Lievertz, M.D., Indianapolis

Convention

The ISMA's annual convention will be conducted at the Century Center in South Bend, Wednesday through Sunday, Nov. 13-17.

Our Drugwise Salvation Guest Editorial

I find it somewhat amusing to note that the "Wise Men" of last year's state legislature found it so necessary to bring Indiana into the 20th century via their "Drug Substitute Law." Of course, the 20th century has a mere 15 years to go until it is relegated to 19th century status.

It was only such a few years earlier that Indiana, and much of the rest of the USA, was grappling with the problems caused by prescription substitution and "brand name" drug counterfeiting. It was just such problems that

led to the anti-substitution laws that have now been scrapped. What short memories the ward-healer legislators havet

All this legislation has been done under the guise of saving people money. There is no evidence that anyone will save very much, if any, money. If our Indiana taxpayers really want to save money, we should elect a legislature dedicated to meeting once every 10 years or so for not more than a three-day session.

The same concept would yield even greater financial returns if the same format were applied to the "Pirates of the Potomac" institution (the Congress). I could even visualize a near-Utopia situation in which we would elect a state governor every 10 years for one term only. Come to think of it, Congressmen and legislators might be changed to one term only.

The advantages to all this, of course, would be that each of these public servants would be relieved of the natural tendency to base all decisions on the prospect of re-election, a process that may or may not result in good laws. -L. A. Arata, M.D., Shelbyville

Look-Alike and Sound-Alike **Drug Names**

BENJAMIN TEPLITSKY, R. PH. Brooklyn, N.Y.

Look-alike and sound-alike drug names can be misinterpreted by a nurse reading doctors' orders or by a pharmacist compounding physicians' prescriptions. Such misunderstandings can result in the administration of a drug not intended by the prescriber. Awareness of such look-alike and sound-alike drug names can reduce potential errors. Category:

Brand Name:

PROMAZINE Antipsychotic

Sparine, Wyeth

PROMETHAZINE

Antihistamine, Antiemetic

Phenergan, Wyeth Remsed, Endo Promethazine HCl

Tablets, Suppositories, Syrup, Injection

Generic Name: Dosage Forms:

Category:

Brand Name:

Generic Name:

Dosage Forms:

Promazine Tablets, Injection, Syrup, Concentrate

ZANTAC

Zantac, Glaxo or Roche Ranitidine

Tablets

XANAX

Histamine H, antagonist Antianxiety agent Xanax, Upjohn Alprazolam Tablets

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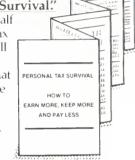
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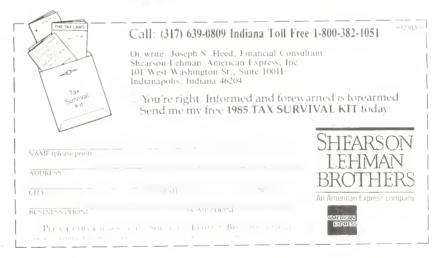
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AUXILIARY REPORT

Muriel Osborne (Mrs. John) ISMA Auxiliary President 1985-86

On April 25, at the ISMA Auxiliary House of Delegates in Vincennes, a new board of directors was installed. This is the 58th year your auxiliary has been privileged to work with the Indiana State Medical Association to promote good health for all people in Indiana.

Your ISMA Auxiliary has four major areas of concern: membership, legislation, health projects, and AMA-ERF.

Membership is basic to any organization. I deem it a challenge that auxiliary membership is only half of ISMA membership. We need to encourage greater increases in the number of our members at large. We must recruit membership among the ever increasing number of female physicians spouses. Retaining members will be a part of our membership program. It is our goal to have all spouses of ISMA officers and chairmen on our membership roster.

Our legislative chairman, Anne Schuster (Mrs. Dwight) from Marion County, brings many years of legislative expertise to her position this year. A legislative newsletter and another day at the capitol will be instituted for auxilians to encourage their active interest in legislative issues affecting medicine now and in the future.

Sylvia Scheeringa (Mrs. Joseph A.) from Allen County is our health projects chairman. Allen County Auxiliary has successfully implemented the Cancer Society's program, "An Early

Welcome

Muriel Osborne, a past president of Delaware-Blackford County Medical Auxiliary, lives in Muncie where her husband, Dr. John Osborne, is a general surgeon in private practice. They have three daughters, two sons, and seven grandchildren.

A member of the ISMA Auxiliary board of directors for the past four years, Muriel has served as AMA-ERF chairman, first vice-president, and president-elect.

Born and raised in Illinois, Muriel was graduated from the University of Wisconsin with a Bachelor of Science degree, majoring in Applied Art.

A member of the Alpha Chapter of Psi Iota Xi, Muriel represented them for three years on the Day Nursery Board and for three years on the United Day Care Center board of directors. A member of the Ball Memorial Hospital Auxiliary, she just completed a three-year term on their board where she served as corresponding secretary, publicity chairman, and vice president.

Start to Good Health," in their grade schools. With financial assistance from the ISMA, Sylvia will develop a brochure to explain this program and its development to our county auxiliaries and members at large. This is an excellent public relations project for the auxiliary.

To enhance ISMA interest in the elderly segment of our society, your auxiliary will promote the AMA Auxiliary television and radio public service announcements titled, "Don't Retire From Life." Auxilians will be encouraged to use these segments in their local communities with audio and visual credits given to ISMA and ISMAA. Thank you for providing these radio and television tapes to the state auxilians free of charge.

Our state AMA-ERF chairman, Lura Stone (Mrs. Robert) from Noble-La Grange County Auxiliary, encourages counties to sell boxed Christmas cards and develop Holiday Sharing Cards in addition to their many individual fundraising projects for AMA-ERF. Your auxiliary is grateful for your sustained interest and financial contributions to our AMA-ERF fund-raising programs.

Thank you for providing this page each month to communicate the programs and concerns of the ISMA Auxiliary.

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BOOK REVIEWS

Hypnotherapy of Pain in Children with Cancer

By J. R. Hilgard, M.D. and Samuel LeBaron, Ph.D. Copyright 1984, William Kaufmann, Inc., Los Altos, Calif. 249 pages, hardcover, \$18.95.

Hypnotherapy of Pain in Children with Cancer is an interesting book, easy to read. It provides a history of hypnotherapy with simple explanations of its applications and techniques. It centers on a study of the use of hypnotherapy in easing the pain of schoolage children and adolescents undergoing bone marrow aspiration. All of them were being treated for cancer at the oneology unit of the Children's Hospital at Stanford University. The 24 who volunteered for the study were generally those who had experienced more pain and anxiety at the time of their previous bone marrow exams.

Five (21%) of the patients had achieved low scores when tested for hypnotic susceptibility. None of those emerged in the group considered "successful." Nineteen (79%) were judged more hypnotizable and for ten (52%) hypnosis was successful in pain reduction. Thus, the success rate for the 24 volunteers was 42%. In all cases hypnosis was an adjunct to the usual management methods—explanation and local anesthesia.

It is unfortunate that the authors titled their book as they did. The above



"Very good. However, you still have a problem."

study comprises just 13 pages of the book. The discussion of the study takes 28 pages while 42 pages are used for background information about the specific topic. In 13 pages are described nonhypnotic methods of relieving pain, but it is unclear if the anecdotal information included pertains to the study patients. Finally, there are 202 pages of interesting but extraneous material about hypnosis. Two hundred two references are cited, of which 25 seem pertinent to the title. From those references examined, one becomes convinced that better ways of protecting children with cancer from the pain and anxiety of necessary procedures must be found and that the hypnotherapist is working hard to provide them. Hupnotherapy of Pain in Children with Cancer is a well written and well edited description of those efforts and concerns.

Parenthetically, it may be reported that the Department of Child Psychiatry at the Indiana University Medical Center uses techniques of hypnosis and relaxation therapy as adjunct therapy for children with cancer and severe burns. Dr. Judith Campbell states that their evaluations also indicate that about half of the referred children are hypnosis susceptibles and can expect benefit from such treatment.

Thomas J. Conway, M.D. Terre Haute Pediatrics

Springer Publishing has released Health Care of Homeless People. Dr. Philip Brickner and his associates at St. Vincent's Hospital in New York, with the aid of professionals across the country, have produced a stateof-the-art guide to medical treatment and program planning. The care of "Street People" differs from conventional practice as much as tropical medicine differs from a comfortable family practice in an ideal location. Treatment of hypertension, exposure, nutritional deficiencies and diabetes differs greatly in the homeless. Treatment of scabies, lice and tuberculosis is entirely different. Hard cover, 368 pages, \$29.95.

Progress in Clinical and Biological Research: Bladder Cancer

By R. Kuss, S. Koury, L. J. Denis, G. P. Murphy and J. P. Karr. Copyright 1984, Alan R. Liss, Inc., New York.

This symposium has 88 contributors which, in my opinion, may be the "urologic world class record" for one topic such as bladder cancer.

It consists of two volumes published in hard back but with apparently less expensive printed pages inside, which appear more typewritten. Part A covers Pathology, Diagnosis and Surgery while Part B covers Radiation, Local and Systemic Chemotherapy and new treatment modalities. Curiously, each book has exactly 453 pages of text. I cannot think of any aspect of bladder cancer that is excluded. Even the history of bladder cancer in the precystoscopic era warrants a chapter.

There is a place in the urologist's or oncologist's library for such an all inclusive text. I particularly enjoy reading ideas of non-American medical writers and to note the similarities and differences between countries. It is noteworthy that treatment is always in a state of flux and dogma can be fleeting.

The newer concepts of prevention such as the use of dietary Pyridoxine and the retinoic analogue of Vitamin A may point toward the best hope of the future.

Rodney A. Mannion M.D. LaPorte Urological Surgery

Brady Communications announces a new book, Common Simple Emergencies, by Dr. Thomas O. Stair and Dr. Philip M. Buttaravolli. Both are FACEP and are director and chairman of Emergency Medicine, respectively, for Georgetown University Medical Center and Holy Cross Hospital, Silver Spring, Md. The book covers minor non-life threatening problems that bring patients to emergency rooms, but which are usually not taught in school or covered in texts or journals. 306 pages, \$19.95.

Synopsis of Diseases of the Chest

By J. A. Peter Pare and Robert G. Frascr. Copyright 1984, W. B. Saunders Co., Philadelphia. 186 pages, softcover, \$39.50.

This is the third major publication of these two distinguished specialists. Dr. Pare, director of the respiratory division, McGill University School of Medicine: Dr. Fraser, Professor of Diagnostic Radiology, University of Alabama School of Medicine. Their well known volume, Diagnosis of Diseases of the Chest, had two editions, 1970, '78. The second edition was expanded from two to four volumes. Realizing that a less comprehensive text might be more useful to residents and practicing physicans, they have taken four years to reduce the larger work to a single volume, retaining the essential material needed by clinicians, and adding new facts and concepts appearing in the literature since 1978. This has required elimination of some of the very extensive bibliography contained in the four-volume set, but an adequate body of references has been retained.

The work is presented in 19 chapters. The first is devoted to a coverage of the anatomy and physiology of the lung. Every known pulmonary disease is dealt with quite adequately under one of the easily understood chapter groupings in the table of contents. The last (19th) chapter is dedicated to tables of differential diagnosis and "decision trees." In the clinical accounts the usual plan of presentation of etiology. pathogenesis, pathologic characteristics, roentgenographic characteristics and clinical manifestations is followed. The basis for treatment is thus established, but there is no formal discussion of therapy.

The chapter on diseases of the mediastinum may be used as an example of how the material is presented. The authors suggest that the reader should review the account of the anatomic arrangement of the three compartments of the mediastinum in chapter 1. Presumably, films of the

chest, both A-P and lateral, have led to the suspicion of mediastinal disease. If there appears to be mediastinal widening, the most productive next step is a CT scan. In a study cited by the authors, the scan correctly identified normal variants, soft tissue masses or vascular abnormalities as the cause of the widening in 92% of the cases studied. Thus, an invasive diagnostic technique was avoided. Acute mediastinitis most commonly results from perforation of the esophagus due to esophagoscopy or primary carcinoma of the viscus, or direct extension from adjacent soft tissue infections. There is usually retrosternal pain. Chronic mediastinitis is usually due to granulomatous lesions, particularly tuberculosis and histoplasmosis. When there is considerable edema and lymph node enlargement, partial obstruction of the superior vena cava should be suspected. Especially with granulomatous lesions, sclerosing mediastinitis may result.

Pneumomediastinitis is observed most commonly in neonates and young infants. Beyond this age group it is seen mostly in the second and third decades of life.

Mediastinal masses situated in the anterior compartment include thymomas, germinal cell neoplasms, thyroid and parathyroid masses and mesenchymal neoplasms. In the middle compartment the majority of masses are due to lymph node enlargement, extension of neoplasms from the lungs,



"Miss Blake, will you please stop saying, 'Slip out of your clothes and the doctor will handle you next'!"

bronchogenic cysts, mesothelial cysts, hernia through the foramen of Morgagni, dilatation of mediastinal veins and pulmonary artery and of the aorta. The posterior compartment, lying between the pericardium and anterior spine, contains the descending aorta, esophagus, thoracic duct, lower portion of the vagus nerves, and posterior group of lymph nodes. Lesions of any of these structures may cause mediastinal widening. Appropriate diagnostic steps beyond the CT scan may be required. Neurogenic neoplasms and cysts are usually in this compartment.

Having read the narrative account of mediastinal disorders, which is freely illustrated with roentgenogram illustrations, the reader then can go to the final chapter of the book and find a very comprehensive table and "diagnostic tree" in which etiology (if known), symptoms and signs, differential diagnosis, further suggested laboratory procedures, etc. are given for the particular type of mediastinal lesion suspected.

Properly used, this volume should be of great value to practicing physicians as well as students and house officers.

Paul S. Rhoads, M.D. Richmond Internal Medicine

A new, expanded edition of Antimicrobial Prescribing is now available. The second edition has been revised and updated by Burt R. Meyers, M.D., the author. \$14.95.

Thieme-Stratton announces a complete clinically oriented guide to differential diagnosis in neurology. The book, Neurologic Differential Diagnosis, is intended for use by neurologists, general practitioners, students and residents. It consists of a translation of a work by Prof. Mark Mumenthaler of the University of Berne. 192 pages, 59 illustrations, hard-cover, \$27.

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NEWS NOTES.

Cooperative Effort Results in New Business Directory

The Marion County Medical Society's Medicine/Business Coalition is a group of physicians and business representatives who have developed the *Physicians' Desk Company Directory*.

The directory is a roster of local businesses. It lists the address of each, together with the type of business involved. It also contains the names of the contact person(s) with title and direct phone numbers, and a statement as to whether restricted work is available. Restricted work, if available, is useful for returning sick or injured employees to employment earlier than they could return to their regular job.

The directory was developed to further better communications between physicians and businesses.

Approximately 1,500 physicians have received a copy of the directory, which is also being sold to business organizations. More than 275 businesses are cataloged; those not listed may be added in the next edition free of charge.

Monroe County PCB Tests Produce Negative Results

Normal levels of blood fats and liver enzymes have been found in Monroe County residents exposed to polychlorinated biphenyls (PCBs), according to laboratory tests conducted by the Indiana State Board of Health and the U.S. Centers for Disease Control.

The State Board of Health and CDC analyzed blood samples from 108 Monroe County residents who had direct or indirect exposure to PCBs.

"PCB exposure studies conducted in other parts of the country have shown elevated levels of certain liver enzymes and blood fats which may be related to the onset of liver disease," said Dr. Woodrow A. Myers Jr., state health commissioner.

The ISBH and CDC concluded that no clear pattern of clinical disease was found in those tested in Monroe County, Dr. Myers said.

Woman Loses Suit for Unsuccessful Sterilization

An action for negligence in performance of sterilization surgery was barred by the statute of limitations where a patient failed to raise the issue of fraudulent concealment as a defense, an Indiana appellate court has ruled.

A physician performed a laparoscopic bilateral tubal ligation on the patient in October 1978. A second physician ordered further testing and, in October or November, a third physician performed a salpingogram to determine whether the sterilization was successful. Neither evidence of the test results nor that of an effort by the patient to discover them appeared in the record. In about July 1979, the patient became pregnant, discovering the fact four to six weeks later and giving birth in May 1980.

In June 1981, the patient filed a complaint against the physicians and the hospital. The trial court held that the action was barred by the statute of limitations and there was no action for wrongful birth or wrongful conception recognized in the state. The court entered summary judgment for the physicians and hospital.

On appeal, the appellate court said that fraudulent concealment would toll the running of the statute of limitations until either the end of the physician-patient relationship or the discovery of the malpractice or of information that would lead to its discovery by the patient. The court took into consideration the lack of a factual assertion that the physicians had actual knowledge that the procedure was unsuccessful and the normal absence of any formal procedure in termination of a professional relationship.

The patient offered no evidence of contact with any of the physicians after November 1978. She contended, however, that the physicians and hospital had a duty to disclose through June 1979. She cited law to the effect that the physician-patient relationship does not necessarily end at the last consultation and that the subjective view of the patient was a factor to be con-

sidered in determining the relationship.

The court said that the physicians and hospital had established a prima facie case that the last day for the patient to file a claim was November 1980. The court pointed out that the patient was aware of her pregnancy and the failure of the tubal ligation at least by August 1979. There remained approximately 14 months within which an action could have been commenced. The court affirmed the trial court's judgment.—*Spoljaric v. Pangan*, 466 N.E.2d 37 (Ind. Ct. of App., July 10, 1984)

(Courtesy of *The Citation*, Dec. 15, 1984.)

GM Introduces Separate Substance Abuse Coverage

General Motors has established what it believes to be the nation's first separate health care coverage for rehabilitative treatment of individuals who have alcohol and/or drug abuse problems.

The substance abuse coverage, which began April 1, is being administered by Connecticut General Life Insurance Co. It covers GM's 2.1 million U.S. employees, retirees and their dependents.



For the Asking . . .

- · "Menus and Recipes to Lower Cancer Risk" is a new booklet that offers tips on how to lower dietary fat content. Tips include trimming fat from meat, removing skin from poultry, using lowfat cheese, choosing prepared food labeled as "low-fat," using yogurt or imitation sour cream instead of regular sour cream, and using skimmed rather than whole milk. It also features recipes that maintain taste but with one-third less fat. Research indicates that lowering the fat content of a conventional diet by one-third is a big enough dietary change to significantly lower the risk of heart disease and arteriosclerosis, as well as many types of cancer. For a free copy, send a stamped, self-addressed #10 envelope to the American Institute for Cancer Research, Dept. FB, Washington, D.C.
- "Understanding Your Medical Examination" is the title of Public Affairs Pamphlet No. 630. The author, Irvin Block, writes to dispel unwarranted concern about the history and physical examination. He describes what the patient can expect and explains what the doctor might be looking for and why. He concludes, "The purpose of the ritual known as a medical examination is the detection of illness in time to cure or potential illness in time to prevent." The pamphlet costs \$1. Write Public Affairs Committee, 381 Park Avenue South, New York, N.Y. 10016.
- Electronconvulsive Therapy is the subject of a National Institutes of Health consensus development conference to be conducted June 10-12 in the Masur Auditorium in Bethesda, Md. To register or to receive a copy of the report, write Ms. Michele Dillon, Prospect Associates, 2115 E. Jefferson St., Suite 401, Rockville, Md. 20852.
- "Guide for Adult Immunization 1985" is a 132-page book published by the American College of Physicians. \$10 each for one to five copies. \$8 each for six to 20 copies, and \$6.50 each for 21 or more copies. Write ACP Adult Immunization, P.O. Box 7777, RO 325, Philadelphia 19175.

- "Diabetes" is the title of the third film in the HEALTHSCOPE public education film series; it was introduced this spring at the annual session of the American College of Physicians, which produces the HEALTHSCOPE series with the aid of a grant from The Upjohn Company. The film will be released to the general public June 1. For more information, call the ACP at (800) 523-1546.
- "Thromboembolic Disease" is a 56-minute videotape that discusses the prevention, detection and treatment of thromboembolic disease associated with general surgery. Showings may be arranged through Norwich Eaton representatives or by writing Professional Services Dept., Norwich Eaton, 17 Eaton Ave., Norwich, N.Y. 13815.
- Olympus Corporation, manufacturer of microscopes, has reproduced a rare microscopy book originally published in Nurnberg in 1687. "Micrographia Nova" by Johann Franz Griendel was reproduced by facsimile plates from the original. Part of the total edition of 300 will be utilized by sending a complimentary copy to each of the owners of the new Vanox microscopes. A limited number of numbered copies will be for sale at \$200. The Olympus address is 4 Nevada Drive, New Hyde Park, N.Y. 11042.
- · "Health and Safety Aspects of Video Display Terminals" is the title of a 36-page booklet prepared by the American Council on Science and Health. The ACSH reports that rumors of miscarriages and birth defects have been found to be groundless, no effect on vision has been demonstrated, and that complaints of headaches, visual fatigue and backaches are due to inappropriate lighting and poor positioning of the tube and the reader. Reading glasses should be made for the special reading distance involved. The booklet advises against setting rigid standards for the equipment because many technical advances at this time would freeze the technology in an imperfect state. For a complimentary copy, send a self-addressed, stamped (39¢) #10 envelope to VDT Report, ACSH, 47 Maple St., Summit, N.J. 07901.

- Industrial safety is emphasized in four new film videos produced by Allied Corp. "Threat from Beyond" (12 minutes) deals with the importance of using personal protective equipment and uses humor to attract the worker and intensify the message. "Judgment at Noon" (11 minutes) stresses that supervisors and workers who ignore safety procedures and unsafe conditions are injured more often. "On Guard" (11 minutes) reviews the basics of machine guarding. "Middle Managers and Safety Accountability" (13 minutes) stresses that middle managers should be aware of the vital role they play in training. Contact BNA Communications, 9439 Key West Ave., Rockville, Md. 20850 – (301) 948-0540.
- "Cost Containment Checklist" is a nine-page publication released by the AMA. It describes practical ways in which physicians can reduce costs. For a copy, write the Dept. of Health Care Financing and Organization, AMA headquarters, Chicago.

Fish and PCBs Don't Mix

The Indiana State Board of Health has issued an advisory against eating certain fish caught in Lake Michigan.

Targeted by the ISBH are brown trout, carp and lake trout more than 25 inches long. Such fish should not be consumed because they are likely to be contaminated with high levels of polychlorinated biphenyls (PCBs), chlordane, dieldrin and DDT.



"Nothing by mouth for 24 hours, and now you want a urine specimen?"

news notes.

Here and There . . .

Dr. William B. Kleinman of Indianapolis has been awarded the 1985 Sterling Bunnell Traveling Fellowship in Surgery by the American Society for Surgery of the Hand.

Dr. Bradley E. Goff of Indianapolis, Dr. Thomas B. Strawn of Lafayette and Dr. James D. Ulm of Indianapolis have been elected to fellowship in the American College of Surgeons.

Dr. Frank P. Lloyd, president of Methodist Hospital of Indiana, has received the "Minority Business and Professional Achievers" award from the Center for Leadership Development in cooperation with the Indianapolis Chamber of Commerce.

Dr. Polly G. Nicely of Indianapolis, chief physician at the Indiana Women's Prison, has been elected the first chairwoman of the Indianapolis YMCA board of directors.

Dr. Thomas J. Parker is the new chief of the medical staff, Dearborn County Hospital, Lawrenceburg; Dr. Elton Heaton is chief of staff-elect, and Dr. Jose G. Ibanez is secretary-treasurer.

Dr. Charles R. Lyons is the new chief of the medical staff at Wabash County Hospital; Dr. James E. Duncan is vicechief, and Dr. James P. McCann is secretary.

Dr. Robert R. Penkava, an Evansville diagnostic radiologist who is president of the Vanderburgh County Medical Society, discussed the future of health care in the county during a March interview with the *Evansville Press* that was aired on WEVV-Channel 44.





Dr. Guinigundo

Dr. Noli C. Guinigundo is the new president of the medical staff, Fayette Memorial Hospital, Connersville; Dr. Jerome R. Giesting is vice-president, and Dr. Gabor S. Tolnay is secretary.

Dr. Kenneth E. Bobb of Seymour has been appointed to the Committee on Mental Health, American Academy of Family Physicians.

Dr. John W. Klemme of Richmond has been elected to fellowship in the American College of Cardiology.

Dr. Robert D. White, director of the Neonatal Intensive Care Unit at South Bend's Memorial Hospital, addressed members of the Council for the Retarded of St. Joseph County in March.

Dr. John R. Stanley of Muncie discussed "The Infertility Work-up" at the March meeting of the Muncie area Resolve Chapter.

Dr. George R. Small of Greenwood was guest speaker at the March meeting of the Morgan County Support Group for Alzheimer's Disease.

Dr. Idillio Elazegui and Dr. Robert A. Ward, Tell City, discussed heart disease and its treatment during a February health forum sponsored by Perry County Memorial and Deaconess Hospitals; Dr. Janet Rippy and Dr. Thomas E. Rose, Evansville, discussed diagnostic testing and intervention.

Dr. Samuel M. Wentworth of Danville was guest speaker at the March meeting of the Johnson County Chapter, American Diabetes Association.

Dr. David J. Dwyer, president of Riverview Hospital's medical staff, discussed stress in professional and personal life during a March public presentation at the Noblesville hospital.

Dr. Kim A. Volz of Jasper discussed cigarette smoking and lung cancer during a recent presentation to the Jasper Kiwanis Club.



Dr. Langston

Dr. Edward L. Langston of Flora has been appointed to the Committee on Drugs and Devices, American Academy of Family Physicians.

Dr. David W. Haines, a Warsaw physician who is a designated FAA medical examiner, discussed aviation medicine during a recent meeting of the Warsaw Pilots Association.

JAMA Expands Distribution

The Journal of the AMA (JAMA) has expanded its distribution to include Burma, Hong Kong, Indonesia, Malaysia, the Philippines, Singapore, Sri Lanka, Taiwan and Thailand.

The Southeast Asian edition joins other international editions of JAMA now distributed in the People's Republic of China, Japan, France, Switzerland, West Germany, Belgium and Italy. It boosts the worldwide circulation of JAMA to more than 600,000.

New ISMA Members

The following physicians were welcomed in February as new members of the Indiana State Medical Association:

Thomas R. Cartwright, M.D., Indianapolis, pulmonary diseases.

Philip A. Countryman, M.D., Indianapolis, family practice.

Stephen F. Dierdorf, M.D. Indianapolis, anesthesiology.

Vincent Dube, M.D. Huntingburg, anesthesiology.

Roger A. Eggert, M.D., Bluffton, otorhinolaryngology.

Martin R. Farlow, M.D., Indianapolis, neurology.

Pinkus Goldberg, M.D., Indianapolis, allergy.

Felix R. Gozo Jr., M.D., Merrillville, traumatic surgery.

Kendall A. Hadler, M.D., Columbus, anesthesiology.

John J. Herling, M.D., Fort Wayne, family practice.

Koduvathara L. James, M.D., Charlestown, cardiovascular diseases.

Billie Jameson, M.D., Indianapolis, pediatrics.

Theodore L. Jansch, M.D., Decatur, pathology.

James A. Lemons, M.D., Indianapolis, neonatal-perinatal medicine.

Carl K. Matlock, M.D., Anderson, emergency medicine.

Paul L. McHenry, M.D., Indianapolis, cardiovascular diseases.



James E. McKiernan Jr., M.D., New Albany, neurology.

Ronald I. Reisman, M.D., Indianapolis, emergency medicine.

Richard L. Scales, M.D., Indianapolis, diagnostic radiology.

Borys Surawicz, M.D., Indianapolis, cardiovascular diseases.

Hartley M. Thomas, M.D., Crown Point, family practice.

Domenic E. Vinciguerra, M.D., Indianapolis, anesthesiology.

Douglas P. Zipes, M.D., Indianapolis, internal medicine.

Increased Liability Risk in Contracting Explained

The Medical Practice Letter announces a new physician contracting guide.

"The Revised Physician's Contracting Handbook," which is now available, was prepared by the California Medical Association and is based partially on examination of more than 200 contracts being offered to physicians in California. It also contains opinion on liability exposure inherent in contracting as prepared by CMA's legal counsel.

The book, which is not limited to California statutes or case law, discusses fully the increased risk of professional liability incurred in most contracts.

The CMA Dept. of Contact Evaluation/Negotiation Services will provide written analyses of contracts for medical societies outside of California on a fee-for-service basis.

For a copy of "The Revised Physician's Contracting Handbook," send a check for \$10 (payable to Sutter Publications) to Sutter Publications, Inc., 44 Gough St., San Francisco 94103.

- Physician Recognition Awards —



The following ISMA physicians are recent recipients of the AMA's Physician Recognition Award. This award is official documentation of Continuing Medical Education hours earned, and is acceptable proof in most states requiring CME in re-registration that the mandatory hours of CME have been accomplished.



Abeleda, Lamberto V., Shelbyville Alvis, David L., Indianapolis Anderson, James T., Greenfield Brown, Gordon T., Indianapolis Caldwell, Kendall W., Connersville Caylor, Charles H., Bluffton Constan, Evan, Valparaiso Dhar, Sisir K., Terre Haute Diotallevi, Gary H., Newburgh Hamaker, Ronald C., Indianapolis Hibbeln, Frederic P., Indianapolis Karia, Pravin M., Jeffersonville Kephart, Stewart B., Bluffton Linne, Mark T., Bluffton

Liebschutz, Norman H., Indianapolis Livingston, Peter H., Bedford Mason, Lester M., Terre Haute McDonald, Joseph D., Evansville Moores, William B., Indianapolis Moriarty, John R., Indianapolis Mullican, William S., Evansville Nuygen, Chung T., Indianapolis O'Connor, Thomas M., Greenfield Paff, James R., Kokomo Panos, Constantine G., Bluffton Paul, Promila D., Munster Peduk, Maria A., Evansville

Pietz, David G., Bluffton
Pratt, George B., Zionsville
Rea, Ralph L., Greenfield
Roberts, Ronald D., Columbus
Robertson, William C., Chesterton
Senasu, Sunchai, Valparaiso
Smith, Don H., Richmond
Smith, Ray C., Indianapolis
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Wills, Martyn A., South bend
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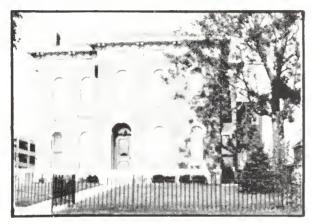
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CME QUIZ.

TO OBTAIN ONE HOUR OF CATEGORY 1 AMA CME CREDIT, answer the following questions by circling the correct answer on the answer sheet below. Complete and clip the application form and mail it to: Indiana University School of Medicine, CME Division, Fesler Hall 224, 1120 South Dr., Indianapolis 16223.

Polycythemia CONTINUED FROM PAGES 367-371

For the following questions choose the best single answer.

- 1. Neonatal polycythemia is seen most frequently in:
 - Premature infants a)
 - h) Appropriate for gestational age infants
 - Large for gestational age infants
- Small for gestational age infants Clinical signs attributable to polycy-
- themia include all of the following except:
 - a) Jitteriness
 - h) Plethora
 - Strong suck e)
 - d) Peripheral cyanosis
- The incidence of polycythemia is alfected by:
 - The site at which the sample is obtained
 - The time at which the sample is obtained
 - The time at which the cord is clamped
 - All of the above
- Which of the following statements is correct?
 - Below a hematocrit of 65% an increase in hematocrit produces a near exponential increase in the viscosity of blood.

- Above a hematocrit of 65% an increase in hematocrit produces a linear increase in the viscosity of blood
- Above a hematocrit of 65% an increase in hematocrit produces a near exponential increase in viscosity of blood.
- None of the above
- 5. Laboratory abnormalities associated with polycythemia include all of the following except:
 - a) Hyperglycemia
 - Hypocalcemia
 - Hypoglycemia e1
 - Transient thrombocytopenia
- The incidence of polycythemia in newborns is approximately:
 - Less than 0.1% of term infants
 - Greater than 10% of premature infants
 - 10-15% of all newborn infants
 - d) 1.5% of all newborn infants
- A potential way to prevent some cases of polycythemia at the time of delivery is:
 - a) Avoid late cord clamping
- b) Administering oxygen to the mother at the time of delivery
- e) Both of the above
- None of the above d)

- When a partial exchange transfusion is performed, an appropriate fluid to exchange for blood is:
 - 4/2 cc of fresh frozen plasma for each 1 cc of blood removed.
 - 1/2 cc of plasma equivalent for each 1 cc of blood removed
 - 1 ce of 1/4 normal saline for each 1 cc of blood removed
 - 1 ce of fresh frozen plasma or plasma equivalent for each 1 cc of blood removed
- 9. Which of the following statements is true?
 - All investigators have found clear evidence that partial exchange transfusions in asymptomatic polycythemic newborns are helpful.
 - Partial exchange transfusions in symptomatic polycythemic newborns are never indicated.
 - The treatment of asymptomatic polycythemic newborns is speculative at this time.
 - None of the above.
- When a partial exchange transfusion is performed, appropriate catheter placement may include any of the following except:
 - Placement of an umbilical venous catheter above the diaphragm or in the umbilical vein
 - Placement of an umbilical venous catheter in the portal vein or one of its branches.
 - Placement of an umbilical arterial catheter at the L.L, inter-
 - All of the above are equally acceptable.

APRIL CME QUIZ Answers

Following are the answers to the CME quiz that appeared in the April 1985 issue: "Bone Marrow Transplantation. . . " by Jan Jansen, M.D., et al.

- 6. b 2. b 7. e 8. d 3. a 4. c 9. a 10. e
- Answer sheet for Quiz: (Polycythemia . . .)

1. a b e d 6. a b c d 2. a b € d 7. a b c d 3. a b c d 8. a b c d 4. a b c d 9. a b c d 5. a b c d 10. a b c d

I wish to apply for one hour of category 1 AMA Continuing Medical Education credit through the I.U. School of Medicine. I have read the article and answered the quiz on the answer sheet above. I understand that my answer sheet will be graded confidentially, at no cost to me, and that notification of my successful completion of the quiz (80% of the questions answered correctly) will be directed to me for my application for the Physician's Recognition Award of the American Medical Association. I also understand that if I do not answer 80% of the questions correctly, I will not be advised of my score but the answers will be published in the next issue of Indiana Medicine.

Name (please print or type)

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To be eligible for this month's quiz, send your completed, signed application before June 10, 1985 to the address appearing at the top of this page.

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OBITUARIES

Edwin V. Marchand, M.D.

Dr. Marchand, 87, a retired Haubstadt general practitioner, died March 10 at an Evansville nursing home

He was a 1922 graduate of Indiana University School of Nursing. He retired from practice in 1973.

Dr. Marchand, a former president of the Gibson County Medical Society, was a member of the ISMA Fifty Year Club.

Gaylord W. Stalter, M.D.

Dr. Stalter, 67, a retired North Webster (Whitley County) general practitioner, died March 20 at his home.

He was a 1944 graduate of Indiana University School of Medicine.

Dr. Stalter began his practice in North Webster in 1947 after completing a tour of duty with the Marine Corps.

James M. Jay, M.D.

Dr. Jay, 55, an Indianapolis internist, died March 23 at his home.

He was a 1956 graduate of Indiana University School of Medicine.

Dr. Jay, who had been involved in medical missions to Zaire several times, was a member of the American Society of Internal Medicine and the American Society of Nephrology.

Malcolm S. Floyd, M.D.

Dr. Floyd, 53, a Good Samaritan Hospital radiologist, died March 8 at his home in Vincennes.

He was a 1956 graduate of the Medical College of South Carolina, Charleston. He was a veteran of two years' service with the U.S. Air Force.

Dr. Floyd, a former treasurer and secretary of the Knox County Medical Society, was certified by the American Board of Radiology. He was a member of the American College of Radiology, the Radiological Society of North America, and the International College of Surgeons.

William H. Clark, M.D.

Dr. Clark, 78, a retired South Bend otolaryngologist, died March 31 of injuries sustained in an automobile accident in Palmdale, Fla.

He received the M.D. degree in 1930 from the University of Mannitoba, Winnipeg. He was an Army veteran of World War II.

Dr. Clark, who retired in 1978, had practiced in South Bend since 1934. In 1968 he was presented the Award of Merit by the Hearing and Speech Center of St. Joseph County. He was a diplomate of the American Board of Otolaryngology and was a member of the American College of Surgeons and the American Academy of Otolaryngology.

Memorials: Indiana Medical Foundation

The Indiana Medical Foundation, Inc. was formed by the Indiana State Medical Association "for religious, charitable, scientific, literary or educational purposes." It provides financial assistance to support the educational mission of Indiana Medicine.

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Karl E. Puterbaugh, M.D.

Dr. Puterbaugh, 88, a retired Albany general practitioner, died Feb. 18 at a nursing home in Muncie.

He was a 1926 graduate of Indiana University School of Medicine. He had practiced in Albany 52 years before retiring in 1978.

Dr. Puterbaugh was honored upon his retirement by having an Albany city park named for him. He was a member of the ISMA Fifty Year Club and the Fifth Year Club of American Medicine.

John Boyd Eviston, M.D.

Dr. Eviston, 91, a retired Huntington general practitioner, died March 18 at his home.

He was a 1929 graduate of Indiana University School of Medicine. He served with the Army National Guard during the Mexican Border uprising in 1916.

Dr. Eviston taught science and coached basketball at Huntington Township and Markle high schools before going to medical school. He was named to the Huntington County Athletic Hall of Fame. He was a member of the ISMA Fifty Year Club.

Charles F. Martin Jr., M.D.

Dr. Martin, 68, a South Bend internist and radiologist, died Feb. 28 while traveling in Florida.

He was a 1942 graduate of Indiana University School of Medicine and was an Army veteran of World War II.

Dr. Martin, who retired in 1983, was certified by the American Boards of Internal Medicine and Radiology, and was a member of the American College of Physicians, American College of Radiology, and the Radiological Society of North America.

COMMERCIAL ANNOUNCEMENTS.

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during the first trimester. Warn patients of the potential
risks to the fetus should the possibility of becoming
pregnant exist while receiving flurazepam. Instruct
patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to
instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nightime sedation. This potential may exist for several days following discontinuation Caution against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, grain ulocytopenia, sweating, flushes, difficulty in locusing, blurred vision, burning eyes, taintness, hypotension, shortness of breath, pruntius, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase, and paradoxical reactions, e.g., excitement, stimulation and hyperactivity

Dosage: Individualize for maximum beneficial effect. Adults: 30 mg usual dosage, 15 mg may suffice in some patients. Elderly or debilitated patients: 15 mg recommended initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.



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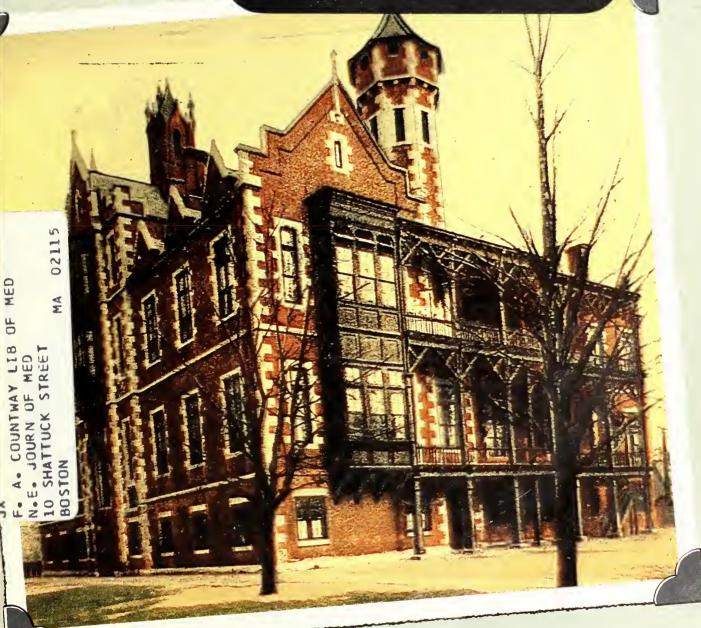
JUNE 1985

VOL.78

NO.6

INDIANA MEDICINE

The Journal of the Indiana State Medical Association



The Original Butter University

1910: A YEAR OF CHANGE, GROWTH, PROGRESS

(See Medical Museum Notes)



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INDIANA

Vol. 78, No. 6 JUNE 1985

Devoted to the interests of the medical profession and public health in Indiana since 1908

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ABOUT THE COVER



Featured is the original Butler University in Indianapolis, which was initially designated as North Western Christian College. The building, razed in 1910, was constructed in 1855 on a 25-acre lot at College Avenue at 13th Street. For more about the school and the people and events of 1910, see Medical Museum Notes on page 444.

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MEDICAL MUSEUM NOTES

CHARLES A. BONSETT, M.D., Indianapolis



people and events of the year 1910, the year of the Flexner report which revolutionized medical education in this country. The year 1910 was one of change and growth and progress. Part of that change is illustrated on the front cover, which shows the original Butler University in Indianapolis (initially designated as North Western Christian College). The building was erected in 1855 at the extreme northeast corner of the city on a 25-acre wooded lot (College Avenue at 13th Street).

The school was unique in being the first college in the nation to admit both sexes on an equal basis. It was here that Dr. Harvey W. Wiley (*J Indiana State Med Assoc*,71:640, 1978) taught Greek and Latin in the late 1860s and early 1870s, and where Dr. David Starr Jordan began his teaching career, although at that time (1875) the school was moved to the Irvington campus, classes there commencing in the fall term (*J Indiana State Med Assoc*, 76:1, 101, 1983).

The building then served for a while as an orphans' home and later as a medical school—the Physio-Medical College of Indiana (*J. Indiana State Med Assoc*, 71:65, 1978). The building was razed in 1910.

Dr. Patrick Henry Jameson (1824-1910), a member of Butler's board of directors for more than 30 years, was the agent for selling the old campus and acquiring the new. He was responsible for an alliance between Butler University and the Medical College of Indiana so that the name of the former appears on the diplomas of the latter until the alliance of the proprietary medical schools to form a state medical school.

Dr. Jameson was a founding member of the Indiana State Medical Society in 1849, and he engaged in a number of civic activities during his long lifetime of practice, which began in Indianapolis in 1843 and continued to within a few weeks of his death



Dr. Jameson

in 1910. Among other activities, he was involved as early as 1899 with six other prominent men in lobbying for a state-supported medical school.

The name of Dr. Patrick Henry Jameson is listed in the 1910 *Who's Who*. He was one of 285 Hoosiers listed for that year. Among others, these well known persons were also included: James Whitcomb Riley, Booth Tarkington, Charles Fairbanks, William Lowe Bryan, George Ade, Gene Stratten Porter, Albert J. Beveridge, Eugene Debs and John Studebaker.

A number of Indiana physicians besides Dr. Jameson were also listed, and most of them were involved with medical education in one way or another. These included:

John F. Barnhill, M.D., a specialist in otorhinolaryngology who was treasurer of the I.U. School of Medicine;

Alembert W. Brayton, M.D. (Indiana Medicine, 77:578, 1984) was a dermatologist, editor, author, professor of dermatology at I.U.S.M. and a past president of the I.S.M.S. (1902);

F. Carrol Heath, M.D. had been associated with the the Central College of Physicians and Surgeons; in 1910 he was a professor of ophthalmology at I.U.S.M;

John N. Hurty, M.D. (*J Indiana State Med Assoc*, 72:217, 299, 1979), commissioner of the Indiana State Board of Health, was a professor of medicine at I.U.S.M.;

John J. Kyle, M.D., a surgeon of the 160th Regiment of Indiana Infantry during the Civil War, was a professor of otorhinolaryngology at I.U.S.M.:

Allison Maxwell, M.D. (*J Indiana State Med Assoc*, 75:499, 1982) was dean of the school of medicine;

Albert Eugene Sterne, M.D. (*J Indiana State Med Assoc*, 70:557, 1977; 73:1, 1980; and 76:441, 1983) was the owner and director of Norways Sanitarium for nervous and mental disease; he was professor of mental and nervous disease at I.U.S.M.

From Muncie there was General William Harrison Kemper, M.D. (*J Indiana State Med Assoc*, 73:424, 561, 1980). Dr. Kemper was president of the I.S.M.S. in 1887 and was identified in the older schools as a professor of medicine and medical history.

Crawfordsville had Dr. William Thomas Gott, a homeopathic physician and surgeon. Richmond had Charles Sumner Bond, M.D., who was (acting) president of the I.S.M.S. in 1894 and president in 1895. Terre Haute had Moses H. Waters, M.D. and Rushville had John Chase Sexton, M.D., who was president of the I.S.M.S. in 1899.

Evansville had Edwin Walker, M.D. and Fort Wayne had Miles F. Porter, M.D., who had been a faculty member of the Fort Wayne College of Medicine and in 1910 was a professor of surgery at I.U.S.M. He had been president of the I.S.M.S. in 1896. Bloomington had Burton D. Myers, M.D. of I.U.S.M. at Bloomington, for whom Myers Hall was named.

In all, the list is very impressive.

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Drixoral* Syrup, a new product in the Drixoral antihistamine/decongestant line, was introduced last month by Schering Laboratories as an overthe-counter product available for the physician's recommendation. Drixoral Syrup is a combination of the effective antihistamine—brompheniramine maleate (2 mg) and the decongestant pseudoephedrine sulfate (30 mg) in a non-alcoholic, pleasant wild cherry tasting syrup. It is indicated for relief of nasal congestion due to the common cold and sinusitis.

The Haemonetics Corporation has a fourth generation Blood Recovery System. The Cell Saver® and Autologous Blood Recovery System safely and efficiently recovers and reinfuses the patient's own red cells. Thus eliminated are disease transmission, transfusion reaction and alloimmunization and conservation of critical blood bank supplies.

Mead Johnson will distribute "b-CapsaTM I Vaccine" (Haemophilus b polysaccharide vaccine). It is the first vaccine designed to protect young children against serious and potentially life-threatening infections. The vaccine is manufactured by Praxis and will be distributed exclusively by Mead Johnson. It is recommended for all children 24 months old and older and children 18 months and older in high risk groups, such as those attending day care centers.

DataChem, Inc. is introducing a new clinical analyzer that performs a variety of blood chemistries, blood coagulation timing, and sodium/ potassium electrolyte measurements. The APSARA Analyzer is operated by a computer. The results are displayed in numeries or in a graphic form. It is fundamentally suited for use in an office or clinic. The computer may be expanded for patient information, billing, insurance, office management and word processing. The APSARA system can communicate with other computers using a modem.

Endo-Lase announces FDA approval for use of the Endo-Lase Infrared Coagulator in the treatment of first and second degree and particularly bleeding hemorrhoids. It coagulates tissue through infrared radiation.

In October 1984, the Practice Builder Ad Agency became the first ever, full-service advertising agency exclusively serving professional practitioners. The specialized agency was developed to provide marketing, advertising, promotion, and public relations services solely for the professional.

Geigy is introducing a new aerosol inhaler that will aid patients who have difficulty using conventional inhalers. Called BrethancerTM (spacer-inhaler), it is a collapsible extension tube about five inches long when extended, into which is placed an aerosol inhaler canister. The longer tube provides the patient with more time to inhale the medication after it is dispensed from the canister, thus requiring less coordination.

Pall Biomedical Products is introducing a new extracorporeal blood filter for use during open-heart surgery. It contains a self-venting membrane, which provides effective and automatic venting of inadvertent air boluses, surges or entrained microbubbles at flow rates up to 6 liters/minute. A final barrier consists of a pleated polyester screen that acts to remove any remaining particulates and emboli larger than 40 microns.

News of what is new in the medical supply industry is composed of abstracts from news releases by book publishers and manufacturers of pharmaceuticals, clinical laboratory supplies, instruments and surgical appliances. Each item is published as news and does not necessarily constitute an endorsement of a product or recommendation for its use by Indiana Medicine or by the Indiana State Medical Association.

Ross Laboratories announces the availability of ROSS SLDTM Surgical Liquid Diet, a highly fortified, juicelike supplement, for patients restricted to clear liquid diets. ROSS SLD provides calories and other nutrients that are deficient in the traditional clear liquid diet. Eight hundred forty calories, 45 grams of protein, and 100% of the US RDA for vitamins and minerals in 1200 mL. It is low residue, lactose free and gluten free.

Hewlett-Packard is offering a five-year guarantee on a new family of defibrillators. Three members of this family offer a variety of usefulness in any hospital area, in general floor situations and in intensive care units. Weights vary from 18 to 23 pounds and are easily portable. A major feature is a fast-charge (two hours) maintenance-free battery which increases reliability and decreases downtime.

Eastman Kodak Company announces Kodak Medlink data management network for clinical chemistry laboratories and hospitals. The Medlink network links Kodak Ektachem analyzers and other automated analyzers with a computer or network of computers. It enables clinical chemistry laboratories to perform on-line quality control functions, automated test and patient data acquisition, total work flow management, work-list and collection-list production, label preparation, billing operations, user-configurable display and report formats.

The Health Care Products Division of U.S. Borax has a new, highly effective antiseptic hand and body cleanser that can be used anywhere in a hospital except surgery scrub. Lurosep® is formulated especially for medical workers who need a safe, gentle, effective hand and body cleanser that can be used repeatedly throughout the day without irritation or toxicity. It contains PCMX (para-chloro-meta-xylenol), which is effective against gram-positive and gramnegative bacteria, yeasts and fungi.

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FUTURE FILE

Welcome for Residents

All ISMA resident physicians are invited to attend an educational program and reception to welcome new residents to the practice of medicine in Indiana. It will begin at 5:30 p.m., Wednesday, June 26, in the Fifth Floor Auditorium of Indiana National bank at One Indiana Square in downtown Indianapolis.

A guest speaker, Dr. Meyer Friedman of San Francisco, will address "The Diagnosis and Modification of Type A Behavior."

For reservations, contact Carol Ann Cunningham at ISMA Headquarters.

Cutaneous Laser Therapy

Harvard Medical School will sponsor a CME course on "Cutaneous Laser Therapy," to be presented Sept. 28 by the Laser Unit of Beth Israel Hospital, Boston.

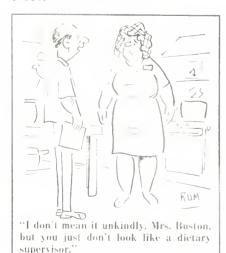
The registration fee is \$150.

To register, write the Harvard Medical School, Dept. of CME, Boston 02115.

Diet and Cancer

The American Cancer Society's second national conference on "Diet, Nutrition and Cancer" will meet Sept. 5-7 at the Shamrock Hilton in Houston.

For information, write the Society at 90 Park Ave., New York, N.Y. 10016.



The Journal of the American Medical Association publishes a list of CME courses for the United States twice yearly. The January listing features courses offered from March through August; the July listing features courses offered from September through February.

Cardiac Morphology

Dr. Bruce F. Waller of Indianapolis will be the program director of a 2½-day program on "Cardiac Morphology: Angiographic, Auscultatory, Echocardiographic and Electrocardiographic Correlations" to be conducted by the American College of Cardiology July 1-3 at the Hyatt Lake Tahoe, Incline Village, Nev.

Contact the ACC, Extramural Programs Dept., 9111 Old Georgetown Road, Bethesda, Md. 20814—(301) 897-5400, ext. 230.

Vascular Surgery

The 15th Annual Peripheral Vascular Disease Symposium, sponsored by St. Anthony Medical Center of Columbus, Ohio, will consider "Critical Issues and Controversies in Vascular Surgery" when it meets Sept. 11 to 14 at the Hyatt on Capitol Square in Columbus.

The fee for physicians is \$450, while the fee for residents, nurses and vascular technologists is \$200. AMA Category 1 credit: 20½ hours.

Contact Shelly J. Hershberger, St. Anthony Medical Center, 1492 E. Broad St., Suite 1100, Columbus, Ohio 43205—(614) 251-3680.

Radiology Courses

The University of California in San Francisco plans four courses in radiology. They vary from two to four days each and are scheduled for early August, mid-October, later in October, and in November. All will be conducted in San Francisco, except the November course which will be held in Maui.

For program copies, fees and other details, write to the University of California, Dept. of Radiology, San Francisco 94143.

Gynecologic Cancer

"Diagnosis and Treatment Strategies for Gynecologic Cancer" is the topic of the 29th annual Clinical Conference sponsored by the University of Texas M.D. Anderson Hospital and Tumor Institute. It will be conducted Nov. 13-16 at the Shamrock Hilton Hotel in Houston.

Contact the Office of Conference Services, M.D. Anderson Hospital and Tumor Institute, 6723 Bertner Ave., Box 131, Houston, Tex. 77030—(713) 792-2222.

Indiana University CME

For the Primary Care Physician
July 23-25-Family Practice Update-Part II, Indianapolis.

Aug. 4-8—Orthopedic Medicine—Part I, Indianapolis. 5-day course, first of 3 parts; led by team of physicians and physical therapists from Europe and U.S.; course deals with examination of joints and treatment of those soft-tissue lesions found in general, sports and industrial medicine. (Also intended for physical therapists, who will be taught deep massage.)

Date negotiable - Mini-Fellowship in Rheumatology. Offered by Rheumatology Div., Dept. of Medicine, I.U. School of Medicine; for general internists, family physicians, general practitioners; 40-hour instructional program at I.U. Medical Center; 5 consecutive days, or other arrangements; program helps attain skills for effective office management of common rheumatologic problems, interpretation of relevant laboratory tests and techniques for joint aspiration and local soft tissue injection; includes experience with "team approach" to management of the arthritic patient. Call K. Brandt, M.D., Rheumatology Div., I.U. School of Medicine - (317) 264-4225.

For the Specialist

July 8-17 — Anatomy and Histopathology of the Head and Neck and Temporal Bone, Indianapolis.

For more information, contact the CME Division, I.U. School of Medicine – (317) 264-8353.

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CANCER CORNER

WILLIAM M. DUGAN, JR., M.D. Clinical Oncology Center, Methodist Hospital of Indiana

1986 Fellowship Grant Program

The Little Red Door, Marion County Cancer Society, is accepting applications for its 1986 Fellowship Grant Program. Each year the agency awards one clinical or research fellowship grant for \$15,000 to a qualified graduate student working in the cancer field.

The goals of the fellowship grant program are to better prepare a physician to work in the field of cancer and to, subsequently, improve the medical care of central Indiana cancer patients.

Linda S. Evans, M.D., a Martinsville resident, is the recipient of the 1985 Fellowship Grant.

Under the direction of the Divisions of Pediatric Infectious Diseases and Hematology/Oncology at the James Whitcomb Riley Hospital for Children, Dr. Evans will receive the specialized training required for treating infections that complicate cancer and its treatment. This training program is the first of its kind in Indiana.

The deadline for submitting 1986 Fellowship Grant applications is October I. Fifteen copies of each application must be supplied, so that the agency's Grant Review Committee may evaluate the requests and recommend their selection to the board of directors. Upon approval of the board, the recipient will be notified by December 1. The grant is for a 12-month period and the money will be provided in January.

For more information about the Little Red Door Fellowship Grant, contact Paul M. Bain, Executive Director, Little Red Door, Marion County Cancer Society, Inc., 1801 N. Meridian St., Indianapolis 46202. The (elephone number is 317-925-5595.

Hoosier Oncology Group

The next meeting of the Hoosier Oncology Group will be an all-day session on Saturday, July 27 from 9:00 a.m to 4:30 p.m. It will be held at Christian Theological Seminary, 1000 W. 42nd St., Indianapolis.

A definite agenda was not available at publication; however, the morning

topic will be "Anti-Emeties," which will be of particular interest to Oncology nurses. There will be an educational session in the afternoon.

For further information, contact Sandra Turner, (317) 630-8927.

Clinical Research Endangered

The Association of Community Cancer Centers (ACCC) points out that clinical research is endangered without adequate reimbursement for the patient care costs. In an article in the Journal of the American Medical Association (Vol. 253, #5, February 1, 1985, pages 684-685) entitled, "The Need for Diagnosis-Related Group 471: Protection For Clinical Research," John W. Yarbro, M.D., Ph.D., and Lee E. Mortenson, M.S. presented data on the higher costs of cancer patient care for those patients on clinical research trials. This makes sense since these patients must receive extra tests and frequently stay in hospitals for longer periods because of the more difficult therapy we give them.

Hospital administrators, concerned over the extra costs and the fixed payment of Diagnosis Related Group (DRG) reimbursement, are less and less inclined to support clinical research at a significant loss to the hospital. The upshot is that clinical research, funded by the National Institutes of Health, may decline significantly over the next few years. ACCC has already seen hospital administrators return research grants to the NIH.

Soon researchers will begin to look at only those problems that hospitals will allow them to do. Then, our clinical research programs will only look at potential new cures that are cheaper, ignoring those that may be better if they are more expensive than the current DRG. The problem has already begun. At one hospital, a researcher was told that an NIH-approved clinical trial was simply too expensive to perform, regardless of its potential or effectiveness.

Over the past 10 years, ACCC has been working with the NIH and Congress to assure that this nation's citizens have available within their own home communities, the latest advances in cancer patient management. But all of this is changing with the advent of DRGs.

When the ACCC first brought this program to the attention of Congress, Congress passed an amendment to the Social Security Act of 1983 empowering the secretary of DHHS to exempt community and university hospitals involved in cancer research from DRGs. The secretary and the administrator of the Health Care Financing Administration (HCFA) determined to exempt only five institutions across the nation . . . hardly capable of managing the more than 20,000 cancer patients annually on clinical research trials.

Congressional leaders suggested that ACCC work with the HCFA leadership to assure that this problem did not continue. The response of DHHS to this problem is to claim that they have never legally supported the costs of cancer patients on clinical trials and will not provide any extra funding now. Indeed, Medicare has paid for these extra costs of patients on clinical trials through its usual cost reimbursement system . . . and is willing to pay for patient care costs now . . . all the way up to the prevailing DRG cap.

Obviously, without adequate funding the costs of clinical research will quickly become prohibitive. Dr. Carolyn Davis, in her response to the *JAMA* article, essentially stated that her hands are tied . . . until Congress passes a law allowing her to do what she did before.

Thus, we need Congressional support for a more specific legislative act that will direct HCFA to pay for those patient care costs of those patients on NIH-approved clinical research trials. Without this law, five years from now we will find clinical research has been set back a decade. ACCC believes that a separate category for approved clinical research reimbursement is the answer, a DRG 471. The article by Yarbro and Mortenson provides some of the details.



PUBLIC HEALTH NOTES

Sickle cell disease affects one in 400 black Americans, constituting a public health concern for the black community. One per cent of black couples in the United States is at risk of giving birth to a child with sickle cell disease. Individuals of Mediterranean ancestry also have an increased chance of having sickle cell trait or disease, as well as thalassemias. In Indiana, an estimated 21 infants per year are born with sickle cell disease.

Manifestations of sickle cell disease are widely variable. Although signs and symptoms of sickle cell generally are not present before the age of three months, hemolytic jaundice has been reported in several neonates with the disease. Where newborn screening is not practiced, all infants at risk for the disease should be tested by four to six months of age. Manifestations of the disease include pain in the joints, bones or viscera, lethargy, pallor or jaundice, fever, infections, splenomegaly, impaired growth and delayed puberty. Strokes, leg ulcers, destruction of spleen tissue and kidney failure can also occur in sickle cell disease. Some individuals with the disease have few significant problems. Intelligence is generally not impaired by siekle cell disease.

Comprehensive care for the patient with sickle cell disease can reduce complications and morbidity and aid in the family's management of the stresses accompanying the disease. Early diagnosis allows education about the importance of preventive care, what problems to anticipate and what level of care is appropriate for different manifestations of the disease.

Most of the medical care for children with sickle cell disease can be provided in an outpatient setting. The family can learn when emergency room visits are needed and when home care is more appropriate. Comprehensive care for the child or adult with sickle cell disease should include routine medical and dental care, hematologic evaluations and regular ophthalmologie and orthopedic

examinations.

As the life expectancy of individuals with sickle cell increases with better medical care, more women with the disease are bearing children. Increased perinatal mortality has been reported in infants of mothers with sickle cell disease.

A couple whose child has sickle cell disease usually has no family history of the disease. Because earriers of sickle cell trait (hemoglobin AS) have no symptoms, they are likely to learn of their carrier status either if they have an affected child or if they have carrier testing.

Genetic counseling allows parents to learn about the choices available to them. These include: a) accepting the risk of having an affected child, b) choosing to avoid the risk by not having children, e) choosing prenatal diagnostic testing during their pregnancies. When prenatal diagnosis is performed, 75% of couples are given reassurance that their baby does not have sickle cell disease. The remaining 25% of couples learning that their baby is affected with the disease may choose to continue the pregnancy, preparing themselves and their families during the remainder of the pregnancy, or to terminate the pregnancy, wishing to spare their child suffering. This choice remains with the family.

Approximately one in 10 black Americans carries sickle cell trait. Testing for sickle cell trait is available without charge through the efforts of several programs in Indiana. The statewide Sickle Cell Anemia Advisory Committee oversees the Indiana Sickle Cell Anemia Program, which is administered by the Indiana State Board of Health. This program is funded by the state and provides screening, counseling and education in three major metropolitan areas (Gary, Indianapolis and South Bend) where approximately 73% of the state's black population resides. Funding for this program will reach \$156,625 in fiscal year 1985-86 for the three grantees: the Northwest Indiana Sickle Cell Foundation, Inc., the Indianapolis Sickle Cell Center, and the Urban League of South Bend and St. Joseph County.

Ten states currently have newborn screening programs which include sickle cell and other hemoglobinopathies. In five of these programs, all infants are screened, while in the other states, only "at-risk" infants are screened. Indiana is the newest of these: In the 1985 Indiana legislature, a bill was passed which authorizes screening for six disorders, including the hemoglobinopathies. This law will become effective July 1, 1985.

Newborn screening will allow the identification of infants with sickle cell trait and sickle cell disease so that parents can be counseled. For parents of infants with sickle cell disease, the importance of early and comprehensive medical care can be stressed. Other family members can be offered testing and counseling, as they are more likely to have the trait than the general population.

The Indiana State Board of Health will sponsor a workshop, "Issues in Sickle Cell Screening, Counseling, and Management," Friday, June 14, at the State Board of Health, 1330 W. Michigan St., Indianapolis. A keynote presentation will be made by Arthur Provisor, M.D., director of the sickle cell referral program at James Whitcomb Riley Children's Hospital, Indianapolis. More information on the workshop can be obtained from John Meaney, Ph.D., chief of the Genetics Section at the State Board of Health, 317/633-0805.

(The Sickle Cell Branch of the U.S. Dept. of Health and Human Services has recently published a guide for the management and therapy of patients with sickle cell disease. Copies can be obtained through the Genetic Diseases Section of the Div. of Maternal and Child Health, Indiana State Board of Health.)

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What to Do About Medicare

A Message from the Executive Director

EDICARE IS THE LARGEST health financing program in the United States; and except for Social Security retirement benefits, could be considered the largest entitlement program in the federal budget. Current outlays are estimated at \$62.7 billion in F.Y. 84, \$71.8 billion in F.Y. 85, and \$77.2 billion in F.Y. 86.

The Reagan Administration is seeking \$3.9 billion in Medicare spending reductions this year. Although Congress is unlikely to adopt all of the Administration's specific cost-cutting proposals, it is also looking for ways to cut a program that is heading for bankruptey.

Under the current system, Medicare Part A (Hospital Insurance) is funded by a payroll tax which now totals 2.6%. Rates are scheduled to rise to 2.9% of payroll in 1986 with another rise to 5.08% scheduled by 1995. This is becoming a considerable tax bite and yet the actuaries say it is not enough to maintain the fiscal solveney of the Hospital Insurance Trust Fund beyond 1997.

On the other hand, Part B or Supplementary Medical Insurance (SMI) requires the payment of a monthly premium by beneficiaries (currently \$15,50). This premium is adjusted annually so that it covers 25% of Part B costs. The remainder of SMI outlays are financed from general revenues.

Under current law, those eligible for coverage under Part A (H.I.) pay no premium at all, even though the average benefits they receive are much greater than their contributions during their working years.

When the Congressional Budget Office (CBO) submitted its annual report



DONALD F. FOY Executive Director Indiana State Medical Assn.

on federal budgetary options to Congress in February 1985, it suggested among other measures, instituting an income-related premium for Medicare Part B (SMI). Several influential members of Congress, including Senator Robert Dole, Senator David Durenberger, and Representative Edward Madigan, have also expressed interest in tying the amount of the Part B premium to income. A White House policy group also recently suggested raising the Part B premium for higher income beneficiaries as well as taxing Medicare benefits as income.

The present rather calm reception

for these ideas provides a dramatic contrast to the furor that was generated back in 1982 when the Office of Management and Budget (OMB) first floated the idea of applying a means test to Medicare. While the present ideas are less far-reaching, they could pose a challenge—to—Medicare's—basic philosophy.

One of the primary concerns in evaluating means-testing as an option for Medicare is the question of whether the Medicare program is to be viewed as a benefit (entitlement) or social insurance program. Most experts would argue that Medicare, unlike Medicaid, was conceived and developed as a social insurance program rather than as a welfare benefit program. However, the structure and particularly the financing of Medicare implies that it may not be purely a social insurance program. Contributions to the Hospital Insurance Trust Fund (Part A) have only been made since 1966, and yet the payment of benefits to the aged and disabled far outstrip the actuarial value of their contributions into the system. Moreover, the level of contributions made is not tied directly to the amount of benefits received.

Part B (SMI) has even less claim to being social insurance. It receives no payroll tax contributions as does the Hospital Insurance Trust Fund, and any elderly person can participate regardless of Social Security eligibility. As mentioned earlier, SMI (Part B) premiums currently pay for only 25% of program costs so it could be considered largely a benefit program.

Since Medicare beneficiaries under Part B pay monthly premiums of \$15.50, which is only 25% of program costs with the balance coming from general revenues, why should not Congress consider having Medicare enrollees bear a greater degree of program costs and alleviate the burden placed on general tax revenues? Assuming that actual Part B costs can be offset by premiums of \$62 per month ($$15.50 \times 4$), why not construct an income-related premium? For instance, we know that at least 20% of Medicare enrollees have annual incomes in excess of \$30,000 and that incomes of the elderly are growing rapidly. If only the 20% of enrollees with incomes of \$30,000 per year and above paid a premium pegged to actual Part B program costs (\$62/month), this would generate additional income to the program of \$372 million annually. As it is now, Part B (SMI) has even less claim to being social insurance than Part A (HI) since it receives no payroll tax contributions and any elderly person can participate regardless of Social Security eligibility.

I believe that if at least Part B Medicare were restructured to reduce federal outlays from general revenue, considering beneficiary income could distribute the added burden on enrollees in such a way as to lessen the impact on those with low incomes. This would pose less of a threat to their

access to eare than new across-theboard requirements or restrictions.

We are currently experiencing Corporate America's reaction to Medicare's prospective reimbursement system (DRGs) in the form of the growth of alternative delivery systems. The development of HMOs, CMPs, PPOs, and ASCs is designed to offset the cost shifting anticipated from DRGs to the private sector.

However, DRGs should make hospital eosts more predictable. With such predictability it would seem that inpatient care under Medicare Part A could lend itself to an income-related premium. As a matter of fact, liberal Johns Hopkins University economist, Karen Davis, has proposed combining Medicare Parts A and B into a single program financed by a combination of existing payroll taxes, general revenues, and an income-related premium administered through the tax system. To me this makes a great deal of sense and is one of the recommendations I had made back in 1983. At that time there was great reluctance to even talk about means-testing under Medicare. However, I believe attitudes have changed recently with the realization that means-testing should not be dismissed out-of-hand; that indeed something such as this may be necessary to save the program.

To take the great burden off the Hospital Trust Fund, which is in serious trouble, Congress should consider requiring beneficiaries to pay a premium for Part A coverage similar at least to what is presently required under Part B (\$15.50 per month). Again, if only the 20% of beneficiaries with annual incomes in excess of \$30,000 were required to pay a token premium comparable to what is paid under Part B, this would produce additional revenue of \$93 million annually to the hospital trust fund without being a great financial burden to anyone.

Time is running out for dealing with the fiscal solvency of the program. The "band-aid" approach of the past only buys time - and a short time at that! Congress will have to face the problem and take some meaningful action soon. Fundamental changes in the program must be made within the next several years in order to insure the viability of the program for those of us who must continue to contribute to its support. Otherwise, if there is no Medicare program, or one that is severely restricted in benefits when the present generation becomes eligible, there will be a large number of very unhappy people.

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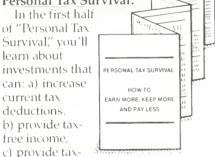
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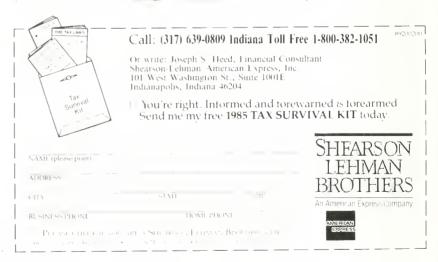
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To obtain Category 1 credit for this month's article, complete the quiz on page 531.



Acquired Immunodeficiency Syndrome: An Overview

ROBERT L. BAKER M.D. KENNETH H. FIFE, M.D. ROBERT A. McDOUGAL, M.D. TIDS IS HERE in Indiana, and many physicians have or will be seeing new cases in the near future. It is our task to become familiar with the syndrome so that we may know how best to treat these patients and to deal with undue misunderstanding among paramedical personnel caring for them.

The acquired immunodeficiency syndrome (AIDS) was first reported in June 1981, when an unusual incidence of *Pneumocystis carinii* pneumonia and Kaposi's sarcoma was noted in homosexual patients. Since then, numerous cases have been reported. The Center for Disease Control (CDC) has defined AIDS as a reliably diagnosed disease at least moderately predictive of a defect in cell-mediated immunity, occurring in a person with no known cause for diminished resistance. The most noted complications have continued to be

Pneumocystis carinii pneumonia and Kaposi's sarcoma. Others are listed in Table 1.1

The AIDS epidemic has aroused national interest and concern. While the majority of cases have occurred on the east and west coasts, Indiana cases also have been reported. The effects of AIDS are far-reaching, and concern among high-risk groups is understandable. The many enigmas, the severity of the disease, and the explosion of information about AIDS have resulted in misunderstanding and undue fear among medical personnel and lay persons associated with AIDS patients. Research interest is intense, with benefits potentially beyond the immediate gains in understanding AIDS and extending into advances in immunology, virology, oncology and their interrelations. The impact on society as a whole is potentially tremendous, and

From the Indiana AIDS Task Force: Indiana University Hospitals, Community Hospital and Hendricks County Hospital—Depts. of Medicine, Division of Infectious Disease, and Pathology.

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the economic demands may be par ticularly burdensome to some institutions caring for these patients. Some Indiana patients have had hospital bills over \$40,000. The purpose of this review is to acquaint Indiana physicians with what is known about the epidemiology, pathogenesis, clinical features, therapy and prevention of AIDS.

Epidemiology

As of February 11, 1985, 8,218 cases of AIDS had been reported in the United States. Over 80% of the patients have been reported since January 1983. New cases are reported daily, and 40,000 additional patients are expected in the next two years. The majority have occurred in individuals belonging to one of six distinet groups: Sexually active homosexual and bisexual men with multiple sexual partners (72.8%), abusers of intravenous drugs (17.2%), Haitians (3.6%), transfusion recipients (1.2%), steady heterosexual partners of AIDS patients (0.8%), and persons with hemophilia (.6%). Overall, 90% are males, while 90% of patients who acquired the disease from heterosexual contact are females. All females have been heterosexual. The majority of patients are between the ages of 20 and 40 years. Seventy-two patients have been less than 13 years of age. Cases have been reported in 45 states, Puerto Rico, and the District of Columbia. Seventy-five per cent of the cases have been residents of New York. California, Florida, or New Jersey with 40% occurring in New York City. Thirty-four patients have been reported in Indiana and 12 have been from Marion County (Charles L. Barrett, M.D., Indiana State Board of Health). Cases have been reported from France, Germany, the United Kingdom, other European countries, Canada, South America and Africa.

The pattern of transmission of AIDS is similar to that of Hepatitis B. It appears that the AIDS agent is transmitted through breaks in the mucosa of the rectum or vagina, or

TABLE I Infections/Complications of AIDS

- 1. VIRAL
 - *Cytomegalovirus
 disseminated
 pneumonitis
 retinitis
 encephalitis
 colitis
 adrenalitis
 viremia
 - *Herpes simplex
 persistent unusual
 disseminated
 Herpes zoster
 Progressive multifocal
 leukoencephalopathy
- 2. BACTERIAL
 - *Mycobacterium arium-intracelluare

disseminated bacteremia

*Mycobacterium, others lung

liver other

- *Nocardia Legionella
- 3. FUNGAL
 - *Candida albicans
 oral thrush
 esophagitis
 disseminated
 - *Cryptococcus neoformans disseminated meningitis pneumonitis
- * (Seen in Indiana cases)

- *Histoplasma capsulatum disseminated Coccidioides immitis progressive pulmonary disseminated *Asperaillus
- Petriellidium boydii 4. PROTOZOAN
 - *Pneumocyst is carinii
 pneumonitis
 retinitis
 - *Toxoplasma gondii disseminated pulmonary encephalitis Cryptospovidium Isospora belli
- enteritis 5. HELMINTHS
- Strongyloides stercoralis
- 6. NEOPLASTIC

 *Kaposi's sarcoma
 cutaneous
 lymph nodes
 gastrointestinal
 oropharynx
 other
 Lymphoma
 - Squamous carcinoma tongue
 - Clocogenic carcinoma rectum
- 7. OTHER
 2Autoimmune thromboeytopenic purpura, anemia

by exposure to blood from transfusions or sharing of needles or razors. Among homosexuals, a higher incidence of AIDS is seen with multiple sexual partners and activities leading to mucosal trauma with exchange of semen and blood.

Transfusion-associated AIDS cases include 53 adults, and an additional 13 cases in children. The CDC has completed its investigation in 13 adults and seven pediatric cases; in 12 of the 13 adult cases the agency

has identified at least one donor who was either in a group known to be at high risk for AIDS, or who had an abnormality in T-lymphocytes. Ninety AIDS patients have been identified who have donated blood within the past five years (CCBC Newsletter, June 1984). As of October 15, 1984, the total number of hemophiliac patients with AIDS was 52. Most of the hemophiliacs with AIDS had been treated with Factor VIII concentrates.

Overall, AIDS mortality has been 48%, including 73% among those diagnosed before January 1983. Mortality is highest among those patients with both *Pneumocystis carinii* pneumonia and Kaposi's sarcoma. Of the 34 reported Indiana cases, 18 have died. (Charles L. Barrett, M.D., Indiana State Board of Health).

Pathogenesis

The infectious complications seen in AIDS patients are primarily those associated with a defect in cell-mediated immunity. These immunologic defects appear to account for the majority of complications seen in AIDS patients. In general, patients with AIDS have decreased numbers of circulating lymphocytes. When specific populations are examined, the reduction in lymphocytes represents a loss of T-lymphocytes and a loss of the helper category of T-lymphocytes. Despite the rough correlation between T-cell numbers and degree of immunosuppression, these numbers are still not predictive, and individuals with profoundly decreased numbers of helper T-lymphocytes and profoundly abnormal helper/suppressor T-cell ratios may still remain free of opportunistic infections. Most AIDS patients do have in vivo correlates of these in vitro T-lymphocyte abnormalities and are broadly anergie and will not react to cutaneous skin tests to common antigens such as mumps or candida. This skin test anergy can also be quantitated in the laboratory by looking at lymphocyte reactions to specific stimulation by antigens.

Abnormalities in the humoral branch of the immune system mediated by B-lymphocytes have also been documented. Many authors feel that these changes are secondary to the changes in the T-cell system and are not truly primary immunologic abnormalities. Nevertheless, there are a number of abnormalities which can be detected, including hyperglobulinemia and polyclonal B-cell activa-

June 1985

tion. Despite this increase in circulating antibodies, these patients appear to be relatively unable to make new antibodies to previously unseen antigen. Thus, although the humoral arm of the immune system seems to be continuously stimulated, it appears to be unable to respond normally to an immunologic challenge. Despite this defect, AIDS patients do not appear to be unusually susceptible to encapuslated bacteria, commonly seen in patients with defects in humoral immunity.

Etiology

When the disease was first diagnosed, many possible etiologies were entertained. These ranged from infectious agents to a number of potential toxins. However, as information about the disease began to accumulate, the bulk of evidence favored an infectious etiology with a transmission pattern that paralleled other viral diseases such as Hepatitis B.

Although a number of previously recognized viral agents, including Cytomegalovirus (CMV) and Epstein-Barr virus (EBV) have been examined as possible etiologic agents, no consistent association has been demonstrated. In 1980, workers at the National Cancer Institute reported the identification of a new virus associated with cutaneous T-cell malignancies. This virus was called human T-cell leukemia virus (HTLV) and was shown to be a typical retrovirus. Retroviruses have been known for years to cause cancer in animals, but no human retrovirus has been isolated prior to this.

One interesting feature of HTLV was its apparent ability to infect human T-lymphocytes selectively. Because of this property and the fact that AIDS was felt to be primarily a disease of T-lymphocyte function, evidence for infection by this newly recognized virus was sought in AIDS patients. Early reports suggested that antibodies to HTLV could be identified in up to 30% of AIDS patients.

Although these numbers were not impressive, HTLV antibodies were rare in apparently healthy homosexuals. This was, thus, the first agent which appeared to be selectively enhanced in AIDS patients.

Subsequently, workers in France were able to isolate a virus from lymphoid tissue from a patient with chronic generalized lymphadenopathy who was in a risk group for AIDS.4 This virus was a typical human retrovirus which was found to selectively infect human Tlymphocytes, and was termed "lymphadenopathy associated virus" (LAV). At about the same time, workers at the National Cancer Institute isolated a virus from lymphocytes from an AIDS patient and also identified this as a human retrovirus.10 It was found to be closely related to the HTLV which had been previously isolated by that laboratory, but did have some differences.

All together, three different types of HTLV have so far been identified. The original virus isolated is now termed HTLV I. This is a virus that was used in the initial screening studies of AIDS patients. The virus isolated from AIDS patients has been termed HTLV III. Many workers suspeet that HTLV III and the virus called LAV are, in fact, the same virus but comparative studies have not yet been completed. Studies have now been conducted looking at percentages of patients with antibodies to HTLV III in groups at high risk for AIDS." In the initial report, 88% of all AIDS patients were found to have antibody to HTLV III and over three-fourths of the patients with the syndrome felt to be a precursor of AIDS were also found to have antibodies to HTLV III. Although antibodies to HTLV III were also found in approximately one-fourth of apparently healthy homosexual men, the majority of the latter individuals had additional risk factors for AIDS, such as many sexual partners or excessive rectal trauma. Antibodies to HTLV III were found in less than 1% of the general population. Additional studies are currently underway to better define the frequency and scope of HTLV III infection in various populations.

Besides the serologic studies which implicated HTLV in the etiology of AIDS, there are additional biologic studies which are also compatible with this hypothesis. HTLV III has been found to be highly selective for infection of human helper T-lymphocytes. These lymphocytes in culture are gradually killed by the virus over a period of time following infection. This m vitro behavior strongly re sembles the defect seen in AIDS patients; that is, a selective loss of helper T-lymphocytes. For this reason, it is felt likely that HTLV III is. in fact, the etiologic agent of AIDS.

Clinical Features

The diagnosis of AIDS is based on the recognition of the signs and symptoms of complicating infections and neoplasms. Recognition of AIDSrelated conditions is also important. An enzyme-linked immunosorbent as say is now available to detect antibodies to HLTV III. Members of high risk groups with or without signs and symptoms of AIDS or AIDS-related conditions may have antibodies to HTLV III. The natural course of all HTLV III antibody-positive individuals has not been clearly defined. Occasional HTLV III antibody-negative individuals may have AIDS.

Until more information and diagnostic tools become available, AIDS is a clinical diagnosis. The most prominent clinical features are those of the secondary processes. Incubation, prodrome and related conditions are less well defined. However, many patients experience weeks to months of generalized malaise, anorexia, weight loss, intermittent fevers and night sweats, diarrhea, and other nonspecific signs and symptoms prior to diagnosis. In addition, withdrawal, apathy, confusion, and mutism out of proportion to diagnosed complicating diseases can be seen late in the

course. Autoimmune anemias, throm bocytopenias, and tumors other than Kaposi's sarcoma are seen. A syndrome of persistent generalized lymphadenopathy with associated constitutional complaints in homosexuals (the "lymphadenopathy syndrome") has been considered by some a prodrome to AIDS. Benign reactive follicular hyperplasia is seen histologically and recurrent, non-life-threatening infections can be noted to occur in these patients.

While follow-up to date in some series has revealed as high as 17% progression to AIDS," other series have shown no such progression. A convincing argument that the "gay lymph node syndrome" is another manifestation of the AIDS agent has been made.18 It should also be noted that a significant number of patients with pneumocystis pneumonia or Kaposi's sarcoma have had antecedent adenopathy, and this trend should not be ignored. For now, recognition of the complicating infectious and neoplastic diseases remains of utmost importance.

Pneumocystis carinii

Pneumocystis carinii is a protozoan capable of causing pneumonitis in immuno-compromised individuals. Pneumonitis occurs in about threequarters of AIDS patients and is the most common opportunistic infection. In contrast to other immunosuppressed children and adults with Pneumocystis carinii pneumonia, the onset is typically more insidious with gradual onset of cough, dyspnea and fever prior to recognition of pneumonia (weeks to months). In addition, arterial hypoxemia is not as profound. Occasionally, routine chest xrays and arterial oxygen tensions are normal. A gallium-67 scan may be useful in picking up occult pneumonitis. Examination of lung parenchyma best establishes the diagnosis, as well as revealing concomitant alternative infection. Imprints of freshly cut lung, as well as fixed specimens stained with methenamine silver, will reveal the cysts. Empiric therapy with trimethoprim and sulfamethoxazole or pentamidine may be suggestive of infection if improvement occurs. However, concomitant infections, poor response, and side effects of medication make confidence in continuing therapy difficult without tissue diagnosis. It is noteworthy that patients with AIDS and Pneumocystis carinii pneumonia experience more side effects, especially rash and leukopenia, when treated with trimethoprim and sulfamethoxazole than do other immunocompromised patients.™

Kaposi's Sarcoma

Kaposi's sarcoma is a multi-centric tumor with vascular structures intertwined in a network of collagen fibers resting among spindle cells. The cell of origin is unknown. An association with CMV infection and HLA DR-5 histocompatability antigen has been noted. Prior to the AIDS epidemic, it was a rare tumor in North America, usually occurring on the lower extremities of elderly men as a localized, nodular tumor, ranging in color from blue to purple.

Among AIDS patients, Kaposi's sarcoma has occurred in approximately 30%. Of patients with Kaposi's sarcoma, almost all (93%) have been homosexual or bisexual.15 These tumors are more aggressive and atypical lesions are usual. Involvement of lymph nodes, oropharynx, and gastrointestinal tract are fairly common, occasionally without skin manifestations. Involvement of the liver, spleen, pancreas, adrenals, lung. brain, testes, aorta, and heart have occurred.16 Again, association with prior CMV infection, and HLA DR-5 antigen has been noted. Patients with HLA DR-5 antigens have lesser degrees of immunodeficiency than those who are HLA DR-5 negative.

Other Infections

A wide variety of other opportun-

istic infections, neoplasms, and autoimmune phenomena have been seen or should be expected in AIDS patients (Table 1). Latent viruses of the herpes group are common, especially CMV and herpes simplex virus (HSV). Unusual organ involvement, such as colitis, encephalitis and adrenalitis, has been seen with CMV. Prolonged, extensive, progressive and atypical lesions of HSV occur. Progressive multifocal leukoencephalopathy (PML) caused by a papovavirus and occurring as a subacute demvelinating disease with progressive weakness and cerebellar dysfunction has also been reported.

Bacterial infections, notably mycobacterial infections, and especially those of the Mycobacterium aviumintracellulare group are noted with pulmonary, CNS, and disseminated disease. M. avium-intracellulare has also been reported in blood cultures.18 A heretofore uncommon infection, it is now no less difficult to eradicate and is resistant to most anti-mycobacterial agents. Investigational agents added to traditional therapy have resulted in some success. Other mycobacterial species, including Mycobacterium tube reulosis as well as other bacteria, such as Legionella species and Nocardia species, have been seen in AIDS patients. Listeria monocutogenes Brucella species, and Salmonella species are to be expected, as they too are dependent on cell-mediated immunity for control.

Fungal infections are common. Oropharyngeal, esophageal and disseminated candida infections are well known. Persistent oropharyngeal candidiasis may predate diagnosis by months. Cryptococcal and aspergillosis infections occur. The region of the country plays a role in which other fungi are seen and are to be expected. For instance, in Indiana, complicating disseminated histoplasmosis occurs and should be considered. In other regions of the country, coccidioidomycosis and blastomycosis should be expected.

Parasitic disease other than Pneu mocystis carinii, most notably toxo plasmosis and cryptosporidiosis, are seen. Both disseminated and central nervous system toxoplasmosis occur and respond to combination pyri methamine and sulfadiazine therapy. Enteritis caused by Cryptosporidium species has been a disease noted in animals and self-limited when occurring in humans. However, AIDS patients experience unrelenting infection which has been poorly responsive to investigational therapies. and most patients have persistent infection to death. In addition, pulmoinvolvement has nary been described.19 A recent study, however, suggests efficacy of spiramycin.3

It should be pointed out that the diagnosis of one opportunistic infection or neoplasm complicating the AIDS syndrome should not exclude other possibilities. To the contrary, multiple infections and neoplasms may occur simultaneously. Indiana patients have been seen to have concomitant Pneumocystis carinii pneumonia, disseminated Mycobacterium arium-intracellulare infection, disseminated histoplasmosis, CMV disease, and herpes simplex virus disease. Evaluations involving tis sues should include analysis for all possibilities. Lack of typical histopathology should not exclude diagnoses. For example, special stains of bone marrow and lung may reveal acid-fast bacillus or organisms compatible with Histoplasma capsulatum without formation of granulomata.

In summary, a variety of heretofore unusual infections and neoplasms should be expected and sought in patients at risk for AIDS.

Treatment

There is, as yet, no known effective treatment for AIDS. With the identification of the presumed etiologic agent of AIDS, treatment directed at, the virus thought to be responsible for the disease could pre-

sumably be developed. None of the currently used anti-viral agents appear to be highly active against any of the retrovirus group. Recently, however, suramin, an anti-parasitic drug, has been shown to inhibit HTLV III reverse transcriptase, block infectivity and prevent viral replication in vitro. It has protected normal helper/inducer T-cells in vitro. The National Institute of Health is conducting a pilot study of safety and efficacy in vivo.

Up to this time, most efforts at treatment of AIDS patients have involved attempts to reconstitute the effective immune system. One substance which has been used in therapeutic trials is interferon. Several different preparations of interferon have been used in an effort to reverse the immunologic abnormalities in AIDS. The interferon used in these situations has been primarily directed at stimulation of the immune system rather than an effort to eradicate the virus causing the disease. Although various interferon preparations have been stimulators of the immune system, the reports received so far on the use in AIDS patients have been unencouraging. There is, however, evidence that interferon has a favorable effect on the course of Kaposi's sarcoma." Several studies are still underway and it will be some time until the final outcome of such studies is known.

The other major compound which has been used to stimulate the immune system is interleukin 2 (IL2). This substance is normally present in small amounts in the body and is a potent stimulator of T-lymphocyte growth. Addition of IL2 to in vitro cultures of T-lymphocytes from AIDS patients has shown an improvement in some of the parameters used to measure T-cell function. For this reason, several centers are attempting to give exogenously administered IL2 to AIDS patients to improve their immunologic function. Once again, the studies are still in the relatively early

stages and the final verdict of this mode of therapy is uncertain. Prelim inary results appear disappointing.

A wide variety of other stimulators have been tried, but in no case have the results been encouraging. There are new chemotherapy regimens being developed for Kaposi's sarcoma which have been reasonably successful in at least partial remissions. However, long-term, tumor-free survival has yet to be documented with any of these chemotherapeutic regimens, and relapses appear to be relatively common.

The major therapeutic mode available to AIDS patients is control of the infectious and neoplastic complications of the disease once they arise. The mainstay of this type of supportive therapy appears to be early identification of patients at risk for this disease and aggressive pursuit of specific pathogens. When patients of AIDS risk groups are evaluated, even relatively minor sounding complaints must be thoroughly investigated and diagnosis pursued. Unfortunately, many infectious complications of the syndrome respond poorly or not at all to currently available chemotherapy. The future of therapy for AIDS patients lies in further development of anti-viral drugs, such as suramin, which are active against the agent of AIDS.

Prevention

There is essentially no risk to the general public or even to easual contacts of AIDS patients. Prevention centers on high risk groups and falls into three broad categories involving (a) homosexual and bisexual males, (b) individuals exposed to blood and blood products including hemophiliacs, patients requiring transfusion and intravenous drug abusers, and (e) health care workers.

Homosexual and bisexual men are advised to decrease numbers of sexual partners and avoid unknown contacts or contacts with multiple partners, contact with AIDS or AIDS- suspected patients, or contacts who have a history of intravenous drug abuse. In addition, rectal intercourse, especially traumatic, is inadvisable. In general, exchange of blood, semen, urine or feces should be avoided.

Blood product and transfusion related AIDS prevention is directed at excluding transfusion of blood from incubating or AIDS eases. A variety of measures have been used including screening donors for history of drug abuse, symptoms of AIDS, and allowing high risk groups the option of telephoning blood centers following donation to avert use of their blood. Asking sexual preference is not practiced. Designated donor programs have also been rejected. Other measures being used include screening blood for abnormal helper-to-suppressor T-lymphocyte ratios and screening blood for antibody to Hepatitis B core antibody as a surrogate test for AIDS. With the discovery of HTLV III and its antibody, blood banks are now screening for the antibody. However, whether all seronegative individuals are safe and all seropositive individuals are infectious will need to be determined.

Cryoprecipitate is made from a single donation of whole blood from a volunteer donor while factor VIII concentrate is made from pools of 10,000 or more donors, many of whom are paid. Therefore, newly diagnosed, mild or young hemophiliaes (under 4 years) should be begun on cryoprecipitate and not the concentrate. Central Indiana is fortunate in having adequate supplies of cryoprecipitate, Long established hemophiliaes are advised to continue either cryoprecipitate or factor-VIII concentrate as prescribed by their physicians.

Health care workers have little danger of significant exposure. There have been 361 health care workers who were exposed to AIDS and studied by the CDC. These included nurses, physicians, phlebotomists, respiratory therapists and other

health eare workers. Most exposures occurred in direct patient care areas. laboratories, operating rooms or morgues; these involved needle stick injuries, cuts with sharp instruments and contamination of open skin lesions with potentially infectious body fluids. The CDC concluded that the risk of transmission, if any, is small.3 Other studies of hospital personnel with needle stick or other significant exposure to blood from AIDS patients have shown no evidence of transmission of the virus. Recently, 85 hospital employees with nosocomial exposure to AIDS or lymphadenopathy syndrome patients had repeatedly negative assays for HLTV III antibodies during a three-year period of observation. AIDS patients who are chronic Hepatitis B carriers have transmitted Hepatitis B but not AIDS in needlestick accidents.

The recommended isolation procedure for this disease is blood and body fluid preeautions, the same precautions used for patients with Hepatitis B. Employees exposed to patients with AIDS should use caution when handling blood or bloodsoaked articles and take special care to avoid needle stick injuries. Other bodily fluids, including feces, especially if gastrointestinal bleeding occurs, should be handled with gloves. Health care workers who use common sense in handling blood and bodily fluids have no significant risk for acquiring AIDS.21

Summary

AIDS is here in Indiana and the number of cases will likely increase with time. The severity of the disease has broad medical, social and economic implications. New information becomes available almost daily. We must continue to meet the challenge to provide optimal care for our patients at risk.

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PATIENTS NEEDED FOR DIABETES RESEARCH STUDY

The Diabetes Research and Training Center, Indiana University School of Medicine, is seeking patients for studies with an experimental aldose reductase inhibitor to determine if this drug will prevent or retard the development of diabetic retinopathy. Aldose reductase inhibitors work by preventing the accumulation of sorbital in tissues including the lens, nerves and retina. They have already been found to prevent metabolic cataracts and to improve diabetic neuropathy in experimental trials. Since the retinal cells accumulate sorbital, this trial is designed to determine if the administration of the drug would prevent retinopathy or retard its progress. The drug is ex-

perimental and although no serious side effects have been found, this study will require close follow-up for one to two years. The potential benefit for patients would be close follow up of their diabetes and retinopathy including retinal photographs and the possible prevention of a serious and disabling complication of diabetes. The trial is in a double-blind format. Otherwise healthy patients with either type I (juvenile) or type II (adult) diabetes are being sought. Patients either without retinopathy or with non-proliferative retinopathy would qualify. Patient referral may be made by calling or having the patients call the Diabetes Center at (317) 630-6374.

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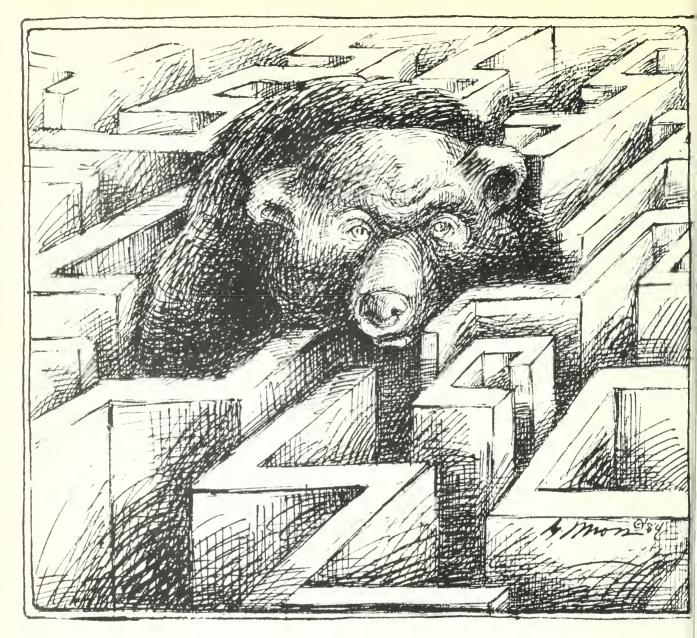
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Ultrasound Augmentation of Central Nervous System Tumor Therapy

ROBERT F. HEIMBURGER, M.D. Indianapolis

Abstract

Ultrasound has been used to treat 20 patients with malignant central nervous system tumors during the past 13 years. Three different modalities of ultrasound have been used. High intensity focused ultrasound was used for 13 patients in an attempt to destroy tumor tissue. Low frequency medium intensity ultrasound was used in two, to determine if the skull could be penetrated to produce local hyperthermia in the neoplastic tissue,

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Acknowledgments: This work was made possible through the generous support of the late R. O'B P. Fortune and his family ... Professors F. J. Fry and R. C. Eggleton designed the high intensity focused and low frequency ultrasound instruments used in this study. They were also involved in each lesioning session with the high intensity focused ultrasound instrument . . Doctors T. D. Franklin and J. T. Patrick also spent much time in the treatment of the patients reported here. . . . Misses Karen Lucas and Yann-Fenn Wu typed and retyped the manuscript. . . . The Medical Illustrations Departments at Indiana University School of Medicine and Chang Gung Memorial Hospital produced the photographic materials. . . . My thanks to all of these.

and benefit conventional therapies. In two others this modality was used with one or both of the other modalities. Low intensity nonfocused ultrasound was used to increase penetration of chemotherapeutic agents into the tumor cells, exclusively in three patients, and in combination with or after one of the other modalities in an additional four.

The diversity of neoplastic conditions treated, and ultrasound modalities used, does not permit a statistical analysis of results, except that the

malignant gliomas were benefited significantly more than the other malignant tumors treated, including metastatic lesions. The apparent prolongation of duration as well as quality of life suggests that investigation of ultrasound therapy for malignant gliomas be continued, and a statistically valid sampling made.

Key Words: Glioblastoma Multiforme, Malignant Neoplasms, Ultrasound, Chemotherapy, Stereotactic Guidance, Augmentation

■ROM 1938 TO 1949, ultrasound was used in Japan and Germany for treatment of maligprimarily cutaneous melanomas.8, 11, 12, 13, 23 Many patients had significant benefit. In 1949, the "Erlangen Resolution,"4, 11, 28 after hearing several reports of obvious increase in tumor growth rate, prohibited the use of ultrasound therapy for malignancy except in experimental animals. Subsequent to that resolution there have been many improvements in the understanding and control of ultrasound energy, as well as the physiological effects produced.

These improvements suggested that this energy form be investigated again in the treatment of neoplasms that have proven to be unresponsive to other therapies. Demonstration of accurately controlled high intensity focused ultrasound lesions in the brain and spinal cord of animals by Fry, et al,^{5, 7} and in humans by Meyers, et al,^{19, 20} suggested that the technic developed by them be applied to malignant intracranial neoplasms.

Additional animal and human studies were reported by other investigators in the 1960s and '70s.24, 27

Methods

High intensity focus ultrasound was applied to malignant intracranial neoplasms through the intact scalp which covered an acoustic window (large cranial bone flap replaced by a stainless steel wire mesh). A stereotactically guided ultrasound beam was directed into the tumor, which was visualized ultrasonically. Both coronal and horizontal two dimensional scans were made 1 centimeter apart. The outline of the tumor and surrounding anatomical structure could be clearly seen in these scans. 5, 6, 9, 10 The focused ultrasound beam could be accurately directed to various portions of the tumor visualized in these scans.5, 7, 9, 10

Usually, the targets were selected in the growing edge of the neoplasm, where the most energy sensitive cells were presumed to be located. The intensity and duration of exposure for each ultrasound lesion was found from animal studies to produce an area of destruction about $3 \times 3 \times 4$ mm. The lesions could be clearly visualized in ultrasound scans immediately after they were produced, (Fig. 1).

The lesions were so small, and tumor growth so rapid at the time of death that the lesions were never clearly identified in human autopsy specimens. Six to as many as 455 lesions were produced at each lesioning session, with a usual dose of 60 to 150 lesions. Each lesion required 0.4 to 0.6 seconds to produce. The computer-controlled stereotactic ultrasound instrument moved the focal point of the ultrasound beam from one target, preselected on the two dimensional ultrasound images, to the next in a few seconds. Forty to 60 targets were selected on each two dimensional ultrasound image. One to six ultrasound tomographic images, scanned 1 centimeter apart, were used in planning a single treatment.

From animal data it has been determined that the possible additive effect of each ultrasound lesion persists for less than two seconds. For this reason the fear of accumulative damage, due to repeated use of the destructive ultrasound beams, was not a deterent to multiple lesion production, as has been the case with X-radiation. Many of the patients were able to ambulate into the ultrasound treatment theater, and after completion of the lesioning procedure, ambulate out again. Many were treated without hospitalization.

Each patient included in this report received 1 to 10 ultrasound lesioning treatments. Ultrasound lesioning procedures were carried out at varying intervals depending on the patient's neurological and physical condition, and evidence of tumor growth. Neurological deterioration and/or ultrasonic imaging evidence of more rapid tumor growth prompted more frequent ultrasound treatments. These were carried out as often as twice a week and as infrequently as once in

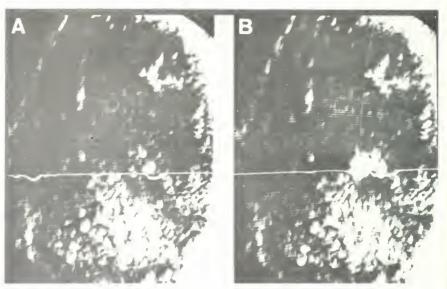


FIGURE 1: Coronal two dimensional ultrasound scans of a human brain containing an inferior parietal Glbm (Table 1, #3). A before and B a few seconds after a high intensity focused ultrasound lesion was made in the superior edge of the tumor, where the cross hairs intersect. The falx can be seen angulating from superior to inferior in the upper left portion of each scan.

two years for patients enjoying a remission of symptoms.

Low Frequency, mid-intensity, ultrasound was used as the only ultrasound therapy in three patients (Table 1, #6, #16, #17). This modality was also used in one (#3) who received all three ultrasound modalities during his 11-year survival with glioblastoma multiforme. Another patient (#18) received this mode of therapy in preparation for low intensity treatments, making a total of five patients.

Low frequency ultrasound of 0.5 to 0.8 Megahertz (Mhz) has been found to penetrate the human skull with less loss of intensity than 1.0 Mhz or higher frequency sound beams (Eggleton RC, ct al: Transbone ultrasonic visualization of the human brain. Abstract 23rd ACEMB, 1974). The greater penetration of low frequency ultrasound was used in an attempt to overcome the need to remove large portions of the cranial vault in order to permit the ultrasound to reach deep brain structures accurately. It was also found to heat deep tissues more

effectively than higher frequency beams. This heating was sufficient to require extra care in protecting the skin from being burned during ultrasound therapy. As a result this modality was not used as extensively as the other two modalities.

To apply low frequency, mid-intensity ultrasound, the scalp was shaved over both temporal areas and covered with a layer of heavy mineral oil or coupling gel. The face of the transducer was covered by a thin plastic membrane containing degassed water to a depth of 1 cm to dissipate the heat produced. An intensity of 0.5 watt/ cm2 for five minutes with a frequency of 0.750 Mhz was used. Each shaved temporal area was usually treated for five minutes during each session. Ultrasound treatments were scheduled in series of six to 10 every few months, depending on the patient's neurological deterioration or lack of it.

Low intensity, 1 Mhz, non-focused ultrasound has been shown to increase blood flow, increase capillary

TABLE 1

Мо	Date	Age	Sex	Tumor		Ultrasound			Survival in Wks after			
				Туре	Site	Mode	Sessions	Total Lesions	lst	Ist	2nd	
								or applications	Sympt	Surg	Surg	Coma •
				634	R't	High		24 117	53	49	31	6
l_	5-03-69	55	M	Glbm	Temp R't	Int	2	34 HI	53	49	31	0
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	10-00-03	1		CA Edily	L't	HI,MI		6 MI		<u> </u>		
3	4-28-70	29	н	Glbm	Temp	LĪ	2	6 HI, 120 L1	708	604	568	1
					R't							
4	5-22-73	28	F	CA Breast	Pariet	HI	2	25 HI	19	15	-	3
_					R't	HI &	_	3.45 .47		١		-
5	1-09-74	29	H	Glbm	Front	LĪ	3	145 HI 0.5 w/cm ²	98	91	51	5
6	1-07-74	39	l m	Glioma	Brain Stem	MI	8	U.5 W/CM² bid 2 Wks	152	17	14	2
0	1-07-74	39		GITORA	L't	шт	· · · · · · · · · · · · · · · · · · ·	DIG 2 WKS	132	1/	14	2
7	4-16-74	59	F	Malig Oligo	Front	LĬ	50	50 LI	717	109	_	6
					R't	HI &	9 HI					
8	5-17-74	49	М	Glbm	Pariet	ŁĪ	LI 160	870 HI, 160 LI	120	109	45	3
	7 05 74		1 _ [Bi-	HI &						
9	7-05-74	44	F	Glbm	Front	LI	3	143 HI, 36 LI	59	51	16	5
10	9-12-74	45	F	G1bm	Front	нІ	5	827 HI	386	364	48	4
	3 12 /4	13	-	G I D III	110110	- 111	,	027 111	300	304	70	7
11	12-03-74	42	H	Glioma	Pons	нї	6	90 HI	260	26	-	3
					R't							
12	12-13-74	3	H	Ependym	Front	HI	5	464 HI	-	138	68	3
12		4.7	l l	61.	Spinal						ĺ	still alive
13	1-14-75	47	M	Glioma Fibrous	T2-3 R't	ΗI	4	20 HI	418	406	-	and active
14	2-24-75	12	м	Xanthoma	Temp	нІ	2	400 HI	_	29	10	2
A -7	2 2 7 7 7 3	12	''	Adireitona	Temp	11.4		400 111			10	-
15	3-17-75	8	м	Glioma	Pons	HI	4	300 HI	52	12	_	18
					R't							
16	8-22-76	38	Н	Glbm	Temp	MI	6	50 MI	208	76	-	4
	0 10 70		_	Recurrent	L't						5.0	7
17	2-13-78	69	F	Accoustic	CPA L't	MI MI &	MI 1	6 MI	108	82	52	7
18	6-06-78	34	F	G1bm	Pariet	LĪ	LI const	MI 7. LI many	312	304	148	6
10	0-00-78			GTDIII	R't	LI &	LI CONST	111 /, LI Hally	312	307	170	still alive
19	5-03-82	38	м	Glbm	Temp	Chemo	36	288 LI	78	72	60	and active
					L't	LI &						still alive
20	10-06-82	62	M	Glbm	Temp	Chemo	24	192 LI	65	60	48	and active

Glbm: Glioblastoma Multiforme. HI: High-intensity focused ultrasound. LI: Low-intensity ultrasound. MI: Low frequency mid-intensity ultrasound.

growth into damaged tissues, and increase passage of chemicals across cell membranes. And the Art Studies made in human brains at autopsy have shown that ultrasound produced by a standard physical therapy unit can pass through the temporal bone and be detected in the ipsilateral brain hemisphere as far as the midline, but not beyond (Heimburger RF and Magill K (unpublished data), 1977).

Low intensity ultrasound with a frequency of 1 Mhz has been used exclusively in one patient (#7), in two patients (#19, #20) to augment chemotherapy, and in a third patient (#18) for two years after low frequency mid-intensity ultrasound had been used to augment her first dose of chemotherapy. This modality was also used for four patients (#3, #5, #8, #9)

in addition to high intensity focused ultrasound lesioning. This makes a total of eight.

Application of this modality was similar to the low frequency, midintensity sound source. As heating was not a serious problem, the water bag was not used, and the face of the transducer was placed directly on the shaved scalp. Nurses and patients' relatives were trained to apply the ultrasound, being sure that sufficient heavy mineral oil or coupling gel was applied to transmit the ultrasound energy through the scalp and skull and into the brain. Ultrasound was applied twice a day for three days after vinblastine sulfate (Velban®) was given intravenously and for four days after each oral dose of lomustin (CCNU[®]). Velban[®] was given weekly in increasing doses as suggested by the manufacturer and CCNU* every five weeks, in repeated series until neurological improvement and symptoms became stable. Chemotherapy and ultrasound therapy was then stopped. Steroids were used throughout as needed.

Patient Selection

All of the patients treated with ultrasound were either starting or well into the terminal phases of their battle with neoplasia, as indicated by headache, lethargy and/or an increase in neurological deficits. Ten had histologically proven glioblastoma multiforme (Glbm). A child (#12) had a rapidly growing malignant ependynoma. Three (#6, #11, #15) were treated for brain stem glioma, and one for a

low cervical high thoracic spinal cord glioma (#13). Two had brain metastasis, one from the lung (#2), the other from the breast (#4). One patient was treated for a huge fibrous xanthoma of the right temporal bone (#14), and one elderly patient (#17) with a large recurrent acoustic neuroma, who was unwilling, as well as too debilitated, for a repeat surgical attempt.

Thirteen patients were treated with high intensity focused ultrasound. Each of the patients treated for Glbm experienced initial benefit, consisting of decreased neurological deficit, decreased headache and improved alertness. This persisted for several months in each case. When the rapid tumor growth spurt started terminally, increased frequency of treatment sessions and number of lesions did not keep up with the rapid cell multiplication, even for the patients who were treated with a large number of ultrasound lesions. When high intensity focused ultrasound failed to provide the initially experienced benefit, four were started on low intensity non-focused ultrasound with the hope of augmenting the effect of the ultrasound destructive lesioning. One (#3) had all three modalities used during his 11 years of survival. Many reviews of his tumor pathology confirmed the diagnosis of Glbm.

In an attempt to overcome the difficulties resulting from removal of a large segment of cranium a low frequency mid-intensity ultrasound instrument was developed, and shown to penetrate the human skull with minimal loss of intensity. It was used briefly for one patient (#3) who had been treated three years earlier with high intensity focused ultrasound, one patient deteriorating rapidly from a brain stem glioma (#6), one debilitated patient as the sole therapy for a recurrent acoustic neuroma (#17), and as the only therapy for one pa tient with a recurrent glioblastoma multiforme (#16).

The one patient (#16) was treated only with low frequency ultrasound

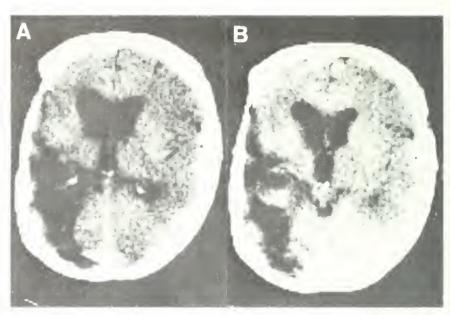


FIGURE 2: Enhanced CT scan of patient #3 with recurrent Glbm in the left parieto-temporal region. A before ultrasound was applied over the lesion. B immediately after the application of Iow intensity non-focused ultrasound at 1.5 watts/1 cm² for five minutes, showing increased enhancement, presumably due to increased permeability of the membranes of the neoplastic cells.

each time deterioration started, after surgery and after radiotherapy. A total of 50 treatments in six separate sessions were administered. He seemed to improve rather dramatically after the first four series of ultrasound treatments until the terminal spurt of rapid tumor growth resulted in his death. Ultrasound when used alone provided no benefit when the tumor started this terminal spurt of rapid growth.

Low intensity non-focused ultrasound delivering up to 3.5 watts per centimeter squared (3.5 w/cm²) at 1 Megahertz (Mhz), from the type of commercially available equipment used in physical therapy, was used through the acoustic window in four patients (#3, #5, #8, #9) who were deteriorating after initial benefit from high intensity focused ultrasound therapy. No chemotherapy or other treatment was used in these patients. These patients claimed to feel better and function better, but there was no apparent decrease in tumor growth rate as observed in the frequent ultrasound, and occasional CT scans, that were made.

Low intensity 1 Mhz ultrasound was used through the shaved scalp and intact temporal bone as the only ultrasound therapy in three patients (#18, #19, #20). It was expected that it would augment the passage of chemotherapeutic agents into the tumor. This hope was backed by animal studies^{13, 16} as well as the demonstration of passage of this type of ultrasound through the intact scalp and skull of humans (Heimburger and Magill (unpublished data), 1977).

Figure 2 shows the CT scans of patient #3 after recurrence of his left temporal Glbm. Scan A is a routine contrast enhanced sean. Scan B shows approximately the same plane immediately after ultrasound had been applied. Exact duplication of the scans was not possible because the patient had to be removed from the scanner to apply the ultrasound and then replaced in the scanner. It appears that

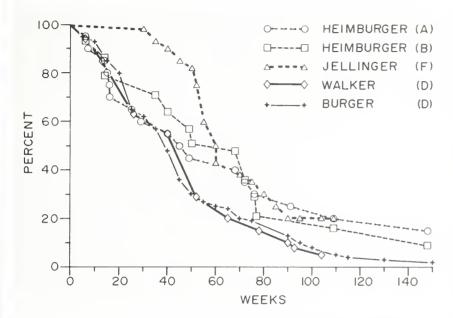


TABLE 2: Graphic comparison of survival of patients with glioblastoma multiforme. Heimburger (A) 13 patients including the one who survived 11 years after diagnosis. Heimburger (B) 12 patients with 11-year survivor excluded. Jellinger (F)¹⁵, Walker (D)²⁶ and Burger (D)⁶ approximations from the graphs of best survival of these three recent authors. The graphs were extrapolated so that all survival figures are recorded in weeks, rather than some in weeks and others in months.

the contrast entered the tumor mass in greater concentration after ultrasound was applied. Additional studies of this finding are underway.

The first of the 3 patients, #18, in which low intensity ultrasound at 1 Mhz was used, was initially started on low frequency medium intensity ultrasound in conjunction with her first doses of chemotherapy. She continued periodic use of low intensity ultrasound at home for two years of good quality life before deterioration prompted a third surgery. She survived the third surgery for 53 weeks, but in a completely dependent state.

Low intensity non-focused ultrasound was started with a combination of Velban[®] and CCNU[®] chemotherapy more than a year ago in two patients (#19, #20) deteriorating after each had had two surgeries with maximal radiotherapy between. Both are still alive and active, many months

longer than expected from available statistical information. (**) 10-20-20-20-

Results

The malignant intracranial gliomas responded much more favorably to ultrasound therapy than any of the other tumors treated in this series. One patient (#13) with a spinal glioma is still alive, and has had significant benefit in neurological functions for 81/2 years following high intensity focused ultrasound therapy. The two metastatic tumors (#2, #4) and the fibrous xanthoma (#14) that were treated appeared to have had no benefit, but also no deleterious effect from high intensity focused ultra sound lesioning. These three patients were deteriorating so rapidly that they received minimal ultrasound therapy, probably insufficient to judge its benefit.

No statistical analysis of the di-

verse conditions and therapies of these patients appears to be appropriate to provide meaningful data. When compared to recent reports of the best results from therapy for glioblastoma multiforme, here, these patients seem to have done as well or slightly better both in duration and quality of life (Tables 1 and 2). The small number of patients in this study, and survival of one patient for 11 years, can skew the conclusions drawn.

Even when the one lone survivor is omitted, this group shows some encouraging increase over expected survival when ultrasound was used to augment or in addition to other therapies (Table 2). Statistically, the malignant gliomas had a longer survival than the non-gliomastous tumors that were treated (p = 0.005). Table 1 lists the patients in order of treatment, the tumors treated and length of survival. A few remained alive but completely dependent (coma) some weeks prior to death. This information is recorded, since survival with poor quality of life is not the goal of tumor therapy.

For the malignant gliomas, ultrasound appears to have prolonged useful life for extra weeks that were not expected from the statistics of other series (*Table 2*). Three of the patients treated (#3, #7 and #10) may be among the small number of patients with glioblastoma multiforme who have an unusually long survival regardless of the therapy used. To offset this group are patients #18, #19 and #20, who appeared to have the more rapidly growing type of glioblastoma multiforme at the time ultrasound therapy was started.

Case #19 had papilledema, left hemiparesis and was severely obtunded when ultrasound therapy was started, in combination with vinblastine sulfate and lomustin chemotherapy. He had had two surgeries with radiotherapy during a span of four months. He is still alive and active, 16 months (68 weeks) after he started chemo-

therapy augmented by ultrasound.

Case #20 was deteriorating rapidly with right hemiparesis and dysphasia less than six months after two surgeries with radiotherapy between. With ultrasound augmenting chemotherapy, this patient has survived an additional 11 months (48 weeks) and is normal neurologically.

The last two patients have had a marked reduction in the abnormalities seen in their CT seans. Midline shift has receded, the areas of attenuation are smaller with a marked decrease in the size and density of contrast enhancement (Fig. 3).

Discussion

The possibility of destroying glioblastoma multiforme with a non-invasive, non-accumulating, accurately controlled energy source like high intensity focused ultrasound seems ideal. The lack of the accumulative effect, which limits x-ray therapy, does not limit the use of high intensity focused, or other modalities, of ultrasound therapy. To offset this advantage, this series of patients has demonstrated that high CSF protein, the product of tumor tissue destroved by excessive use of ultrasound for therapy, is a limiting factor to the indiscriminate use of high intensity focused ultrasound. This limit makes total destruction of the large tumors with high intensity focused ultrasound improbable.

Technology is on the threshold of developing ultrasound beam sources that can penetrate the human skull (Fry, Patrick, Franklin: Transkull ultrasound brain lesioning (unpublished data), 1981). This will eliminate the need to remove a large cranial bone flap, which provided complications in this small series of patients.

The medium intensity low frequency ultrasound used in this study created sufficient heat to make its application hazardous to the scalp, even though penetration of the skull was quite good.

The low intensity ultrasound at 1

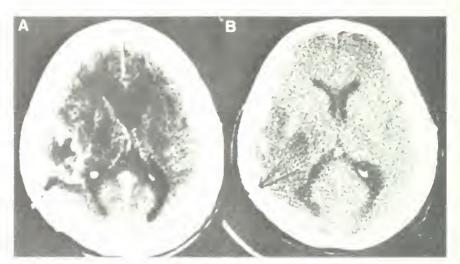


FIGURE 3: CT scan A is of a Glbm in the right hemisphere of patient #19 immediately before ultrasound therapy was started four months after first symptoms. During the prior four months he had had two surgeries and radiation therapy. B, taken six months after chemotherapy augmented by ultrasound was started. Headache, vomiting, lethargy, left hemiparesis and papilledema had subsided. He is still living more than 60 weeks after his second surgery for what appeared to be a very rapidly growing neoplasm. His Karnofsky rating is between 070 and 080.

Mhz used in this study was produced by standard, commercially available, equipment commonly used for physical therapy. It was easily and safely applied. With proper application it was found to penetrate the scalp, skull and meninges to enter the cerebrum of the ipsilateral hemisphere, but was not detected contralateral to the midline (Heimburger and Magill (unpublished data), 1977). Used alone over long periods of time it may have been responsible for increasing tumor growth, due to the increase in blood flow and the capillary growth it encouraged. Accompanied by chemotherapy it appears to have a beneficial effect on gliomastous tumors, particularly the malignant ones.

Ultrasound in this modality appears to be safe and effective when it is used only during the period in which the chemotherapeutic agent is estimated to be circulating in the peripheral blood. This concurs with findings in treatment of transmitted meduloblastoma in hamsters, in which

the tumor was destroyed with ultrasound, but also spread to the lymphatic system, unless chemotherapy accompanied the ultrasound (Fry: Ultrasound and chemotherapy in the treatment of a malignant meduloblastoma model (unpublished data), 1975).

Ultrasound frequency is of crucial importance in its effectiveness for visualizing and treating intracranial neoplasms (Fry, Patrick, Franklin: Transkull ultrasound brain lesioning (unpublished data), 1981). Low frequency ultrasound penetrates the skull well, but produces more heat than higher frequency beams, even when used at the same intensity.

Ultrasound with a frequency higher than 1 Mhz produces less heat, but penetrates less deeply into tissues, does not traverse the adult skull sufficiently to be readily detected. A compromise frequency is therefore required to achieve the desired penetration, without producing excessive surface heat. One Mhz seems to meet the requirements of skull penetra-

tion, producing some but not exces sive heat (Heimburger and Magill (unpublished data), 1977). Additional modification and improvement of ultrasound instrumentation will increase its effectiveness for therapy.

The data gathered in this study, and animal studies, suggest that ultrasound therapy in all the modalities used will be most effective when accompanied by chemotherapy. Studies to find optimal frequencies, intensities and size of ultrasound producing crystals should continue so as to find the best combination that will allow the beams to traverse the skull, and alter neoplastic cells so as to make them more susceptible to chemotherapeutic agents.

Studies that have been done 13, 16, 18, 22, 27 and some currently in progress (Patrick JT: Blood brain barrier changes in an ultrasound field (unpublished data), 1983), show changes in the blood brain barrier in ultrasound fields. They indicate that the blood brain barrier is altered by ultrasound fields that are not of sufficient intensity to produce a visible lesion. 14, 16, 18, 22, 27 These alterations are temporary. The duration has not been completely documented, but appears to be only a few minutes.

Additional studies to more clearly understand the effect of ultrasound on tissues are planned. With the encouragement and knowledge gained from this small series of patients, it seems appropriate to continue to use ultrasound to augment chemotherapy in the management of malignant intracranial and intraspinal neoplasms. A prospective controlled study is under way to determine if the encouragement achieved so far is merely a chance happening or worthy of more extensive study and use.

Careful study of the patients treated with high intensity focused ultrasound suggests that the coneshaped beam passing through the bulk of the tumor to reach its growing edge may have a more beneficial effect than the small area of destruction produced at the focal point. The 100% intensity in the area of focus drops off very rapidly to a nondestructive 80% a few millimeters away. The effect of the less than lesioning ultrasound waves on the bulk of the tumor may provide more benefit than can be accounted for by destruction of a small volume of tumor tissue.

This observation gave the idea for using less intense sound for augmentive therapy rather than trying to destroy the entire tumor mass. With destruction of tumor tissues there was a release of protein into the CSF which rose rapidly over 48 hours to well over 1000 mgm per cent in the two patients (#8 and #10) who received 450 ultrasound lesions during a single lesioning session. The ultrasound lesions themselves caused no immediate increase in neurological signs or symptoms in the two patients. They appeared to be unchanged, except more alert, and carried on their normal activities for six to eight hours after the 450 high intensity focused lesions had been made. They were able to walk, eat, talk and function as they did prior to the ultrasound lesioning.

Six to 8 hours after the ultrasound treatment had been completed, they started to complain of headache, which became increasingly severe with nausea, vomiting and gradually lethargy and papilledema. CSF pressure was not increased, but the protein increased rapidly over the next few days and showed no sign of diminishing for the few days that it was tested. In both instances the patient's condition deteriorated at a greater rate. CT and ultrasound scans showed increase in the size and enhancement of the tumor. Autopsy showed large necrotic tumors without sufficient hemorrhage to produce such a profound change. It seems possible that some immunity factors were altered to decrease host resistance to tumor growth.

In spite of many warnings one of the three patients (#18) receiving low intensity 1 Mhz ultrasound therapy at home continued to treat herself daily for more than two years. The rapid change in her condition suggested that the increased blood flow and capillary growth in the tumor, caused by the ultrasound, produced a more rapid tumor growth. A similar situation was observed in patient #5. These experiences indicate that, in spite of ultrasound being a non-invasive, non-accumulative wave form, it is not completely innocuous and must be treated with an adequate degree of respect. When properly used no increase in neurological deficit, or other changes, have been observed from any of the three ultrasound modalities used in this study.

This series suggests that ultrasound may improve the survival and quality of life for some patients with malignant intracranial neoplasms. Glioblastoma multiforme appears to be more susceptible than the other tumors treated. The two patients (#2 and #4) with metastatic cerebral lesions were not improved. They failed to show even the immediate improvement of function that the patients with gliomas experienced after each ultrasound treatment. Improvement in the feeling of well being that patients with both malignant and pontine gliomas experienced after ultrasound therapy may be attributed to suggestion. The increased motor strength, and coordination, seemed more objective. Because of these improvements, patients with gliomas were eager to repeat the lesioning. Those with metastatic lesions were reluctant.

The experience of this study suggests further investigation of ultrasound to augment the treatment of malignant intracranial neoplasms.

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BALANCED CALCIUM CHANNEL BLOCKADE!



Low incidence of side effects

CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

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Reduces angina attack frequency*

42% to 46% decrease reported in multicenter study.

Increases exercise tolerance*

In Bruce exercise test, control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes (P<.005).

CARDIZEM

(diltiazem HCl)

THE BALANCED
CALCIUM CHANNEL BLOCKER

PROFESSIONAL USE INFORMATION



DESCRIPTION

CARDIZEM* (dittrazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one, 3-(acetyloxy) -5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)monohydrochloride,(+) cis- The chemical structure is

Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform it has a molecular weight of 450.98. Each tablet of CAROIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth

Mechanisms of Action. Although precise mechanisms of its

antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways

1 Angina Due to Coronary Artery Spasm CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial

and subendocardial. Spontaneous and ergonovine-induced cor-onary artery spasm are inhibited by CARDIZEM.

2. Exertional Angina CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal

and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward depolaring current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Dilitazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood

negative inotropic effect. The resultant increases in coronary plood if low (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance. Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, dilitazem decreases sinoatrial and atnoventricular conduction in isolated tissues and has a negative inotropic effect. in isolated preparations in the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diffuziore prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces tolerance studies in patients with ischemic neart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect, cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of dilitazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by dilitazem.

or slightly reduced by dilitazem. Intravenous dilitazem in doses of 20 mg profongs AH conduction

or slightly reduced by dilitazem.

Intravenous dilitazem in doses of 20 mg profongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Oilitazem-associated profongation of the AH interval is not more pronounced in patients with first-degree heart block in patients with sick sinus syndrome, dilitazem significantly prolongs sinus cycle length (up to 50% in some cases). Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Oilitiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bloavailability (compared to intravenous dosinn) of about 40% CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, proprianolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma elevels with 130 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 35 hours. Desacetyl diffuzem is also present in the plasma a levels of following single or multiple drug administration is approximately 35 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary assolitator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given, a 12D-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem

INDICATIONS AND USAGE

Angina Pectoris Due to Coronary Artery Spasm. CARDIZEM

is indicated in the treatment of angina pectors due to coronary artery spasm. CAROIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as

treatment of sportnations coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks)

Chronic Stable Angina (Classic Effort Associated Angina).

CARDIZEM is indicated in the management of chronic stable angina CARDIZEM has been effective in controlled trials in

reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta blockers or of the safety of this combination in patients with impaired ventricular function or conduc

CONTRAINDICATIONS

CAROIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

Cardiac Conduction. CARDIZEM prolongs AV node refrac tory periods without significantly prolonging sinus node recov ery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second-or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem

Congestive Heart Failure. Although diltiazem has a negative

inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.

Hypotension. Oecreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hynotension

Acute Hepatic Injury. In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In ingit tooles of unlazeril were associated with repeate dailage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes;

however, these changes were reversible with continued dosing **Drug Interaction**. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM (See

Controlled and uncontrolled domestic studies suggest that con comitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltrazem has been shown to increase serum digoxin levels up to 20%

Carcinogenesis, Mutagenesis, impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from live to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women, therefore, use CAROIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted

in human milk Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation

Pediatric Use. Safety and effectiveness in children have not heen established

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy. The following represent occurrences observed in clinical studies

which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are edema (2.4%),

headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (asthenia (1.2%), AV block (1.1%). In addition, the following were reported infrequently (less than 1%) with the order of pre tion corresponding to the relative frequency of occurrence

Flushing, arrhythmia, hypotension, bradia, palpitations, congestive heart for Cardiovascular

syncope
Paresthesia, nervousness, somnol

Nervous System temor, insomnia, hallucinations, and am Constipation, dyspepsia, diarrhea, vor mild elevations of alkaline phosphatase, SGPT, and LOH Gastrointestinal

Oermatologic Pruritus, petechiae, urticaria, photosens Polyuria, nocturia.

The following additional experiences have been noted: A patient with Prinzmetal's angina experiencing episor vasospastic angina developed periods of transient asymptomic programme and patient asymptomic programme. asystole approximately five hours after receiving a single dose of CARDIZEM

The following postmarketing events have been reported quently in patients receiving CAROIZEM erythema multiform kopenia, and extreme elevations of alkaline phosphatase, SGPT, LDH, and CPK. However, a definitive cause and effect be these events and CARDIZEM therapy is yet to be established

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been li Single oral doses of 300 mg of CAROIZEM have been well tool by healthy volunteers. In the event of overdosage or exagg response, appropriate supportive measures should be emplo addition to gastric lavage. The following measures may be consi

Administer atropine (0.60 to 1.0 mg), II is no response to vagal blockade, admi Bradycardia isoproterenol cautiously Treat as for bradycardia above. Fixed High-Oegree AV

degree AV block should be treated wit diac pacing Administer inotropic agents (isoprote dopamine, or dobutamine) and diuretics Cardiac Failure

Hypotension Vasopressors (eg. dopamine or levart bitartrate).

Actual treatment and dosage should depend on the severity clinical situation and the judgment and experience of the tr physician

physician
The oral/LO₅₀'s in mice and rats range from 415 to 7401
and from 560 to 810 mg/kg, respectively The intravenous LC
these species were 60 and 38 mg/kg, respectively The oral
dogs is considered to be in excess of 50 mg/kg, while lethali
seen in monkeys at 360 mg/kg. The toxic dose in man is not)
but blood levels in excess of 800 ng/ml have not been asso

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atheroscierotic nary Artery Disease or Angina Pectoris at Rest Due to nary Artery Usease or Angina Pectoris at Hest Due to nary Artery Spasm. Oosage must be adjusted to each pa needs. Starting with 30 mg four times daily, before meals bedtime, dosage should be increased gradually (given in doses three or four times daily) at one- to two-day interval optimum response is obtained. Although individual patient respond to any dosage level, the average optimum dosage appears to be 180 to 240 mg/day There are no available datao ing dosage requirements in patients with impaired renal or function. If the drive must be used in such patients. It trations him. ing dosage requirements in patients with impared renal or function if the drug must be used in such patients, titrationshi carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1 Subilingual NTG may be taken as required to abort anginal attacks during CAROIZEM therapy.

Prophylactic Nitrate Therapy—CAROIZEM may be coadministered with short- and long-acting nitrates, but have been no controlled studies to evaluate the anti-

effectiveness of this combination.

3 Beta-blockers. (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

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Childhood Thoracic Trauma



JOSHUA M. CARESKEY, M.D. Indianapolis

AJOR TRAUMA to the chest wall and intrathoracic viscera is, fortunately, fairly uncommon in children. When such injury does occur, however, there is a serious threat to life. In cases of thoracic trauma associated with significant intracranial and/or abdominal injury, mortality approaches 30%^t. Most deaths occurring early after emergency room arrival and hospital admission are a result of physician failure to recognize, specifically diagnose, or appropriately treat thoracic injury. An appreciation of the clinical and radiographic manifestations of pediatric chest trauma, as well as a working knowledge of the

pathophysiology involved, will, in no uncertain terms, save lives.

The nature of thoracic injuries encountered in the pediatric age group is often related to age. In the neonatal period, the thoracic cavity is susceptible to birth trauma. Resulting from any combination of a) obstetrical manipulations, b) vigorous resuscitation, or c) excessive positive airway pressure, these injuries involve, respectively, fractures (most often of the clavicle), pneumothorax and/or pneumomediastinum, and pulmonary interstitial emphysema.

In the toddler to preschool age, the aspiration of foreign bodies—often a peanut—reaches a peak incidence. This accident can be asymptomatic or even unrecognized for a long period, or it can present as moderate to severe respiratory embarrassment; on occasion, it is fatal. A specific history of a choking episode, or a witnessed aspiration, may not accompany the youngster, necessitating a high index of suspicion in this age group.

Older, school-age children are most frequently the victims of blunt trauma to the chest. The automobile is by far the most common culprit in these injuries. Vertical falls from great heights and violent abuse/battering are causes of injury that appear to be increasing in frequency. Also on the rise is the incidence of penetrating wounds to the chest in teenagers.2 These victims usually reside in volatile inner-city neighborhoods and. with gunshot and stab wounds, present a clinical spectrum not unlike that seen in adults in busy, metropolitan emergency departments.

Diagnosis

When thoracic trauma is obvious or suspected, an aggressive, efficient,

and thorough but brief search must be made to identify a lesion which may be responsible for immediate or impending life-threatening cardiac and/or pulmonary dysfunction. These conditions, representing the so-called "dirty dozen" (Table), absolutely require identification and implementation of therapy immediately thereafter.

Due to the relative elasticity and resiliency of the child's rib cage, blunt trauma generally injures the chest wall little, if at all. A tremendous amount of kinetic energy may be transmitted to the interior, imparting a catastrophc injury to the heart, vascular structures, pulmonary parenchyma, esophagus, vertebral column, and/or spinal cord. The absence of chest wall abrasions, contusions, palpable fractures, crepitus, or other external signs of trauma may provide a false sense of security to the unwarv examiner. Further diagnostic modalities must be undertaken, beginning with auscultation and radiographs of the chest, to rule out serious injuries with a greater degree of certainty.

The sounds of crowing, stridor, and labored breathing must not be overlooked and major obstruction to air flow must be assumed. Coarse, rhonchorous upper airway noises or the opposite, absent breath sounds, alert the physician to a significant airway or intrathoracic problem (see *Table*). Concomitant injury to other organ systems, especially in the context of shock, may further aggravate the child's respiratory status.

In nearly all cases, circumstances will allow time for rapidly obtained x-rays of the chest. If the child's condition permits mobilization, views in the lateral and upright position, as well as the anteroposterior projec-

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tion, may be helpful. A careful search for fractures is made. Inspection for air pockets in the subcutaneous tissue, pleural and/or pericardial cavi ties, and mediastinum may suggest traumatic rupture of the lung, con ducting airways, or esophagus. A shift in the child's extremely mobile mediastinum may signify elevated intrapleural pressure from a tension pneumothorax on the side away from the deviation. Opacification of one or both hemithoraces suggests hemo thorax or well-established contusion, or both. Depression of a hemidiaphragm implies a tension pneumothorax; elevation, with loss of diaphragmatic contour, usually represents a radial tear with possible traumatic herniation of abdominal contents.

Widening of the mediastinum should be considered pathognomonic of heart or great vessel injury until proven otherwise, especially if accompanied by any of the following: 1) inequality of radial pulses; 2) frac ture of the first or second thoracie rib, suggestive of a massive blunt force; 3) hypotension; 4) blood in the left pleural space; 5) left apical "cap ping" on chest films; 6) back pain, and 7) confirmation of the widening on a non-magnified postero-anterior radiograph of the chest. An arch aor togram and possible left ventriculogram should be performed in these cases. The morbidity from an angiogram performed by a skilled radiologist is slight when compared to that from an undiagnosed aortic rupture

Given any of the above signs or symptoms, if one assume the child does not have a vascular catastrophe on the basis of initially stable hemo dynamic measurements and is shown to be correct by continued stability of the patient over two to three days, this does not imply a high degree of clinical acumen. It merely means that the physician (and the patient) "got away with it," were fortunate in a risky assumption and gamble. To re

peat: Any of the above signs or symptoms, in the context of a wid ened mediastinum, represent an absolute indication to invasively study the heart and great vessels. A clinician need not apologize to patient, peers or radiologist for a negative aortogram under these circumstances.

TABLE

Dirty Dozen

(Thoracic injuries capable of producing severe cardiorespiratory decompensation)
Airway Obstruction
Tension Pneumothorax
Flail Chest
Open Pneumothorax (Sucking Chest Wound)
Hemothorax
Cardiac Tamponade
Pulmonary Contusion

Cardiac Injury Great Vessel Injury Traumatic Diaphragmatic Hernia Esophageal Rupture Tracheal or Bronchial Injury

Management

As in other serious trauma settings, placement of a large bore venous catheter for rapid replacement of large fluid volumes is essential following the establishment of a sufficient airway and maintenance of effective alveolar ventilation. To achieve the latter, suctioning of secretions and blood, clearing of loose teeth and debris, and endotracheal intubation are often required. The small size of the oral cavity, the relatively large tongue, the anterior location of the larvnx, and the possible existence of concurrent maxillofacial trauma make visualization of the glottis difficult in the younger child and infant, especially if a cervical spine collar is in place. The most experienced physician in attendance, preferably an anesthesiologist with appreciable pediatric experience, should perform the intubation.

A follow-up chest x-ray is taken to document position of the tube, since the relatively short tracheal length in children promotes bronchial intu

bation, hypoxia, and even a mainstem bronchial perforation. Endotracheal tube internal diameter should be similar to that of the child's little finger or external nares. A formula frequently utilized for tube size selection is 4 plus 1/1 the child's estimated age in years. Smaller tubes are available for the newborn or small infant. If oral endotracheal intubation can not be accomplished, immediate tracheostomy is performed. Surgical cricothyroidotomy is not as easy or as reliable as in the adult and probably should not be undertaken in the smaller patient.

Chest wall injuries, less common in children than in adults, are usually the result of penetrating trauma such as impalement or stab wounds. The sucking chest injury (open pneumothorax) is managed by debridement, coverage with an adherent dressing, and tube thoracostomy away from the injury. Flail chest, again less frequently observed in the pediatric population, may require internal pneumatic stabilization, often for a prolonged period if the flail segment is sufficiently large. 4 External traction on the fractured ribs, once a common treatment modality, is mentioned here only to be condemned."

Up to one-third or more of a child's blood volume may be shed into a pleural space. Hemothorax, from intercostal vessel laceration, internal mammary artery injury, pulmonary laceration, or disruption of the lung hilum is detected by physical examination, x ray, and/or needle thoracentesis. Tube thoracostomy at a low, dependent level is performed to evacuate the hemithorax and quantitate blood loss. Massive or persistent heavy bleeding suggests great vessel injury and is an indication for thoracotomy. Ligation of an intercostal vascular bundle, pulmonary resection, or direct repair of a large vessel may be required. As a general rule, major vascular injury is more common on the left side, and hilar injury on the right.

Tension pneumothorax, manifested clinically by tachypnea, cyanosis, air hunger, absent breath sounds, and a hyperresonant ehest to percussion. can be definitively diagnosed and dramatically relieved by placement of a large bore needle through the second or third intercostal space anteriorly. Following the rapid gush of air, tube thoraeostomy can be performed, even prior to x-ray confirmation. It is believed that death from tension pneumothorax is a result of circulatory, not ventilatory, impairment due to the mobility of the mediastinum and subsequent interference with cardiac filling and emptving.

Contusion of pulmonary parenchyma may be overlooked easily in the emergency room setting, as early chest films often fail to demonstrate the opacity which invariably appears soon thereafter. When contusion, or crush lung, is suspected, initial management is optimized by limitation of infused volumes of crystalloid *after* aggressive management of shock and restoration of a safe circulating plasma volume.

Persistent or massive air leak through the chest tube(s) suggests disruption of the trachea or a major bronchus. Other signs may consist of hemoptysis, pneumopericardium or pneumoperitoneum, subcutaneous emphysema, or atelectasis of the ipsilateral lung. If the patient is stable, bronchoscopy will aid in the diagnosis and localization of the injury, followed by thoracotomy and repair; if unstable, immediate operation is mandatory. When a mainstream bronchus is involved, selective intubation of the contralateral bronchus is performed to maintain oxygenation. On occasion, full cardiopulmonary bypass is required when adequate oxygenation is not otherwise possible.

Blunt forces of great magnitude may rupture the diaphragm, leading to herniation of abdominal viscera (liver if on the right; stomach, spleen, colon and small bowel if left-sided) into the chest and respiratory distress." It is not unusual for diagnosis to be delayed for days or even weeks, though an x-ray demonstrating abnormal diaphragmatic contour or nasogastric tube course should raise the suspicion of this entity. Repair is generally transabdominal since this approach facilitates ease of diaphragmatic repair and allows attention to any associated intraperitoneal injuries.

Traumatic perforation of the esophagus from a blunt force is considered early if pneumomediastinum or left pneumothorax is seen and late if septic shock ensues. Diagnosis is confirmed by esophagoscopy and/or esophagogram. A small leak may be treated by tube thoracostomies and high dose, broad spectrum antibiotics. Larger injuries require right thoracotomy and direct repair. If the extent of damage is uncertain, operation is probably wise. Instances of delayed fistula formation to the airway have occurred, especially when the trachea has also been injured.

Accidents involving the heart should be considered in any patient arriving in extremis in the emergency room, especially if there is a penetrating chest wound. Immediate endotracheal intubation and an open resuscitative thoracotomy to control torrential hemorrhage or relieve pericardial tamponade are necessary. If the patient responds to these measures, transfer to the operating room is made to complete the procedure. This approach has recently been rec-

ommended for victims of penetrating thoracic injury with cardiac arrest and discouraged for those with blunt trauma, especially if there is associ ated severe head injury and absent pupillary reflexes.

Conclusion

Blunt trauma in children will involve the thorax in only about 10% of cases. However, a child with impaired ventilatory mechanics can deteriorate rapidly, making swift, accurate assessment of thoracic injuries in the emergency department of critical importance. Priorities in resuscitation are identical to those in the adult; recognition of the anatomic and physiologic differences in the pediatric patient will allow a more effective management of the smaller victim of trauma.

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Metabolic Alkalosis

Critical Care Medicine

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The Author
Discusses the
Etiologies and
Treatments of
the Most Common
Acid-Base
Abnormality
Found in Hospital
Patients . . .

as a primary rise in the serum bicarbonate associated with an altered serum pH, is the most common acid-base abnormality appearing in the hospital population. It is also probably the most poorly understood of the four cardinal acid-base abnormalities. The purpose of this paper is to refamiliarize the clinician with this perplexing problem's etiologies and subsequent treatments.

In discussing metabolic alkalosis (MA), it must first be understood that the cause of the alkalosis is one process and its maintenance is a second, different process. AA, by definition, must be associated with an elevated serum bicarbonate concentration. This increase is caused through one of three mechanisms: a) loss of hydrogen ion from the extra-cellular fluid (ECF); b) addition of bicarbonate to the ECF (or its equivalent-citrate, acetate, or carbonate); or c) contraction of ECF volume with loss of chloride in excess of bicarbonate.

What makes MA an especially challenging problem, however, is the added concept of maintenance. The MA is maintained by factors that tend to increase hydrogen ion secretion and increase bicarbonate salvage in the kidney. The most potent of these are hypovolemia and hypermineralocorticoidism.

In the normal human, exogenous bicarbonate loading is associated with increased urinary losses of bicarbonate with subsequent return of the serum pH toward normal. This is dependent, however, on the availability

of another cation within the tubular filtrate to replace the excreted bicarbonate, thus assuring continued electrical neutrality in the serum. In the human, the only cation in sufficient quantity to accomplish this is chloride. As the normal kidney excretes an acute load of bicarbonate, hypovolemia may occur via the loss of sodium with bicarbonate. The stimulus to maintain a euvolemic state then initiates normal compensatory mechanisms which override the bicarbonate diuresis through the resultant scavenging of urinary sodium. This process is then manifested by decreased, but not absent, urinary sodium excretion. With this sodium avidity comes the requirement to reclaim a balancing anion. In the absence of a readily available chloride within the tubular filtrate, bicarbonate is absorbed as that anion to preserve electrical neutrality. This cycle, therefore, perpetuates the alkalosis established through the other primary process. This phenomenon is especially apparent in the proximal tubule. In the distal tubule one sees the effects of mineralocorticoids. The hypovolemia stimulates renin which in turn stimulates the release of aldosterone. Aldosterone's effects include the increased excretion of hydrogen ion and potassium in exchange for tubular sodium.3,5 Secretion of hydrogen ion also makes possible the reclamation of bicarbonate via a carbonic anhydrase cycle.

The contribution of hypokalemia to the maintenance of the different types of MAs continues to be a controversial subject. It is known that in most instances hypokalemia accompanies

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MA. It has also been shown, however, that these alkaloses can be corrected without the correction of this potassium deficiency.² Suffice to say, then, that hypokalemia's contribution to MA's maintenance lies mainly in ion shifts and not directly by stimulation of hydrogen ion secretion. This contribution is especially manifest in severe potassium depletion (serum values less than 2 meg/L).⁶

In evaluating MA, the clinician should attempt to define its etiology. The major etiologies of MA are divided into two groups: the chloride responsive and the chloride resistant groups (Table). By definition, and of value diagnostically, the chloride responsive group reduces urinary chlorides to less than 10 mmoles/L while chloride resistant disorders have urinary chlorides greater than 20 mmoles/L. These numbers, of course, assume the absence of renal disease or artificial manipulation of urinary chlorides via loop diuretics, etc. The physician should be alert to the fact that this information (use of diuretics, etc.) may not be volunteered! The third group shown in the Table does not fit neatly into these categories.4

The chloride-responsive MAs are the most common in clinical practice. Nasogastric tube suction and vomiting are two frequently seen etiologies. They have in common the loss of hydrogen ion and chloride from the body and a decrease of ECF volume. Associated hypokalemia occurs primarily through renal losses via aldosterone-induced potassium-secretion while direct gastric loss of potassium is a less important factor. Diuretics (primarily thiazides and loop diuretics) induce a similar MA via the loss from the ECF of relatively bicarbonate-free fluid, increased tubular hydrogen ion secretion, and the contraction of the ECF space with resultant secondary hyperaldosteronism.2.3 Rare causes of this type of MA include cystic fibrosis, congenital chloridorrhea, laxative abuse, or villous adenoma³ and have in common

TABLE

Major Etiologies of Metabolic Alkalosis

Chloride Responsive Metabolic Alkaloses

Gastric-loss (naso-gastric suction/vomiting)

Diuretics (primarily thiazides and loop diuretics)

Cystic fibrosis

Congenital chloridorrhea

Laxative abuse

Villous adenoma

Post-hypercapneic states

Chloride Resistant Metabolic Alkaloses

Cushing's syndrome

Primary aldosteronism

Bartter's syndrome

Adreno-genital syndrome

Mineralocorticoid-like compound (glycyrrhizic acid)

Magnesium deficiency

Unclassified Forms of Metabolic Alkalosis

Citrate loads (blood transfusions)

Milk-alkali syndrome

Acetate loads (intravenous infusions)

Impermeable anions (sulfate, carbenicillin)

Hypo-parathyroid states

Non-parathyroid induced hypercalcemia

the loss of chloride-rich fluids and the potential for hypovolemia. The posthypercapneic state also fits into this group. The compensatory rise in serum bicarbonate in response to elevations in serum pCO2 remains upon normalization of alveolar ventilation and lowering of the pCO₂. This occurs despite the kidneys' attempt to increase bicarbonate excretion. As described earlier such bicarbonaturia may be limited by the availability of a serum chloride ion to be filtered and which may remain low in this disorder2 unless exogenously supplied.

All chloride-resistant MAs have in common states of hypermineralocorticoidism occurring in the absence of hypovolemia. Mineralocorticoids, as discussed above, cause increased secretion of hydrogen ion and potassium in exchange for reabsorbed intratubular sodium. Eventually, a steady state will be reached wherein the secretion of hydrogen ion equals the delivery of bicarbonate plus the

production of metabolic acid (the socalled "escape phase"). This type of MA may be associated with a highrenin state (Bartter's syndrome or magnesium deficiency) or with a low renin-state (primary aldosteronism: Cushing's syndrome, or adreno-genital syndrome). Another less common cause of low-renin/high mineralocorticoid state is the excessive ingestion of licorice. Licorice contains a mineralocorticoid-like compound (glycyrrhizic acid) that, in abundance, can have significant effects on the kidney.2 Differentiation among the possible causes is relatively straight-forward (measurement of renin, physical exam,

There are also various causes of MA in the third unclassified group. Citrate loads (transfusions), milk-alkali syndrome, bicarbonate or acetate (intravenous) infusions all may cause MA via acute alkali loads. These causes of MA require ongoing alkali administration and chloride deficiency (in the patient) to become

chronic. Another rare cause of MA is the administration of an impermeable anion (sulfate, carbenicillin, etc.) which changes the ionic electrochemical potential difference across the tubular cell membrane and facilitates the secretion of potassium and hydrogen ion, thus generating a hypokalemic, hypochloremic MA. Finally, decreased parathyroid hormone secretion and non-parathyroid induced hypercaleemia also are associated with MA.

While we have defined the types and causes of MA, we have not yet discussed the consequences of MA. As the bicarbonate ion concentration rises, the pCO, also rises in accordance with the Henderson-Hasselbach formula. Roughly, for each increase of 1 meg/L of bicarbonate, the pCO. will rise 0.7 mm Hg.78 This relationship holds relatively true throughout the range of bicarbonate rise. It has been shown that alkaloses are associated with increased morbidity and mortality. In addition, central nervous system changes including irritability and seizures can be seen. The decreased ionized calcium concentration associated with alkalosis may be manifest by tetany and a positive Chvostek's sign. Alkalosis can also lead to cardiac irritability with an increased incidence of arrhythmias. Finally the compensatory increase in pCO, mentioned previously can be associated with relative hypoxia secondary to hypoventilation and may lead to respiratory failure.

There is also a frequent association of a *mildly* increased anion gap with MA. The unmeasured anions in the anion gap are primarily plasma proteins and plasma organic acids (phosphate and sulfate). In MA nonbicarbonate buffers (primarily plasma proteins) contribute a hydrogen ion to titrate the developing alkalemia. This titration causes an increase in the net negative charge of the plasma proteins. Secondly, any decrease in ECF volume also increases the concentration of the

plasma proteins. Both of these mechanisms tend to increase the effect of these unmeasured anions thereby increasing the anion gap. The importance of this, to the clinician, is to recognize that an increased anion gap in the presence of MA does not necessarily imply that an unrecognized metabolic acidosis is present.^{11,12}

The treatment of MA must be directed toward the factors causing and maintaining the alkalosis. Factors that increase bicarbonate generation (mineralocorticoids) or increase bicarbonate reabsorption (ECF volume contraction) should be eliminated. Chloride and potassium must be made available to the patient to correct the hypokalemia and hypochloridemia. When a more rapid reversal of the alkalosis is desired, several treatment mechanisms are available which can be tailored to the individual patient. When increased gastric hydrogen and chloride loss occurs, Cimetidine has been used to slow the secretion and subsequent loss of these ions from the body. 1544 The carbonic anhydrase inhibitor, acetazolamide, has been used to increase bicarbonate diuresis. One must be mindful, however, of its kaluretic effect and potential for metabolic acidosis in patients with renal disease.

In the situation where the alkalosis is life-threatening, the clinician has several acidifying agents, or mineral acids, from which to choose. Arginine monohydrochloride has been used in the past but has been associated with hyperkalemia in patients with liver or kidney disease.15 Another acidifying agent, NH₄C1, has been associated with worsened azotemia in renal failure and is contraindicated in patients with liver failure. The mineral acid most frequently used in our institution to reverse MA is hydrochloric acid. This is infused as a 0.1N solution through a central vein to avoid the potential for sclerosis of a peripheral vein. 1641 A recent article has suggested, however, that peripheral venous infusion is possible by

mixing the hydrochloric acid with an amino acid solution and fat emulsion. The hydrochloric acid infusion should not exceed the rate of $0.2 \, \text{mmol/kg/hr.}^3$

The amount of mineral acid required is calculated by assuming the volume of distribution of bicarbonate to be 0.5 times the body weight (in kg)." The formula is:

meq mineral acid required = body wt. (kg) $\times 0.5 \times [\text{observed} \quad HCO_{+}\text{-normal} \quad HCO_{+}]$

During infusion of any of the mineral acids, frequent monitoring of the blood gases, to guard against acidosis, and monitoring of the hematologic profile, for hemolysis, should be done. Evaluation of the infusion site for leakage of the infusate with subsequent tissue necrosis should also be encouraged. Finally, hemodialysis using a high-chloride low acetate dialysate has been especially effective in the setting of chronic renal disease.²⁰

In summary, metabolic alkalosis is a multifactorial abnormality that presents many challenges to the clinician. With a systematic approach to the problem, however, a logical plan of action can be formulated and the alkalosis treated with minimal morbidity.

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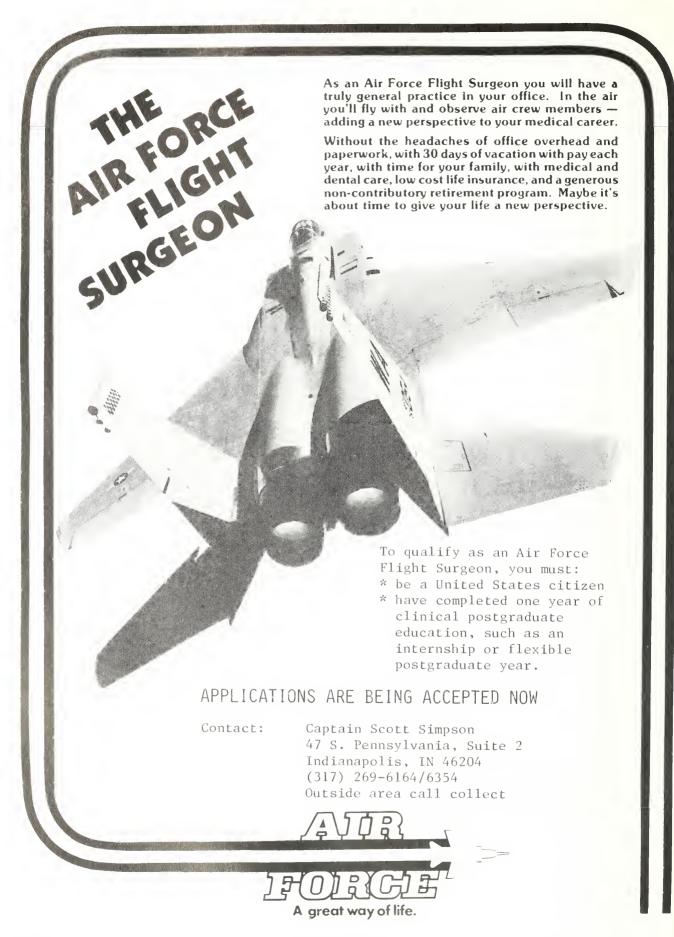
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Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive enal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum obtassium. Hypersensitivity to either component or other sulfonamide-lerived druss.

Varnings: Do not use potassium supplements, dietary or otherwise, unless ypokalemia develops or dietary intake of potassium is markedly impaired. Supplementary potassium is needed, potassium tablets should not be ised. Hyperkalemia can occur, and has been associated with cardiac irreguanties. It is more likely in the severely ill, with urine volume less than the liter/day, the elderly and diabetus with suspected or confirmed renal sufficiency. Periodically, serum K+ levels should be determined. If hyperalemia develops, substitute a thiazide alone, restrict K+ intake Associated widened ORS complex or arrhythmia requires prompt additional herapy. Thiazides cross the placental barrier and appear in cord blood. Ise in pregnancy requires weighing anticipated benefits against possible azards, including fetal or neonatal jaundice, thrombocytopenia, other dverse reactions seen in adults. Thiazides appear and triamterene may pipear in breast milk. If their use is essential, the patient should stop pusing Adequate information on use in children is not available. Sensitivity actions may occur in patients with or without a history of allergy or ronchial asthma. Possible exacerbation or activation of systemic lupus rythematosus has been reported with thiazide duretics.

recautions: The bioavailability of the hydrochlorothiazide component of pazide' is about 50% of the bioavailability of the single entity Theoretially, a patient transferred from the single entities of Dyrenium (tramterene, K&F CO.) and hydrochlorothiazide may show an increase in blood pressure fluid retention. Similarly, it is also possible that the lesser hydro-norothiazide bioavailability could lead to increased serum potassium levels, owever, extensive clinical experience with "Dyazide' suggests that these anditions have not been commonly observed in clinical practice. Do sniodic serum electrolyte determinations (particularly important in patients miting excessively or receiving parenteral fluids, and during concurrent se with amphotericin B or corticosteroids or corticotropin [ACTH]). Specially in the elderly, diabetics or those with suspected or confirmed nal insufficiency. Cumulative effects of the drug may develop in patients the impaired renal function. Thiazides should be used with caution in itients with severe liver disease. Observe regularly for possible blood scrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias we been reported in patients receiving triamterene, and leukopenia, rombocytopenia, agranulocytosis, and aplastic and hemolytic anemia we been reported with thiazides. Thiazides may cause manifestation or tent diabetes mellitus. The effects of oral anticoagulants may be creased when used concurrently with hydrochlorothiazide, dosaga edipicants may be necessary. Clinically insignificant reductions in arterial sponsiveness to norepinephrine have been reported. Thiazides have also sen shown to increase the paralyzing effect of nondepolarizing muscle laxants such as tubocurarine. Triamterene is a weak folic acid antagonist. periodic blood studies in cirrhotics with splenomegaly. Antihypertensive feets may be enhanced in post-sympathectomy patients. Use cautiously surgical patients. Triamterene has been found in renal stones in assolation with the other usual calculus component

lazides may add to or potentiate the action of other antihypertensive Igs.

retics reduce renal clearance of lithium and increase the risk of lithium

verse Reactions: Muscle cramps, weakness, dizziness, headache, dry uth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatical conditions; nausea and vomiting, diarrhea, constipation, other strointestinal disturbances; postural hypotension (may be aggravated by ohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, crus, pancreatitis, xanthopsia and respiratory distress including pneunitis and pulmonary edema, transient blurred vision, sialadenitis, and tigo have occurred with thiazides alone. Triamterene has been found in al stones in association with other usual calculus components. Rare idents of acute interstitial nephritis have been reported. Impotence has an reported in a few patients on 'Dyazide', although a causal relationship in ot been established.

pplied: 'Dyazide' is supptied as a red and white capsule, in bottles of 00 capsules; Single Unit Packages (unit-dose) of 100 (intended for 'lutional use only); in Patient-Pak $^{\infty}$ unit-of-use bottles of 100.

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Cardiovascular contraindications to the use of
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hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome
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and second- or third-degree AV block.

effect is constipation (6.3%).

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Contraindications: Severe left ventricular dysfunction (see Warnings), hypotension (systolic pressure + 90 mm Hg) or cardiogenic shock, sick sinus syndrome (except in patients with a functioning artificial ventricular pacemaker), 2nd- or 3rd-degree AV block Warnings: ISOPTIN should be avoided in patients 2nd- or 3rd-degree AV block **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction < 30% or moderate to severe symptoms of cardiac failure) and in patients with any degree of ventricular dysfunction if they are receiving a beta blocker (See *Precautions*) Patients with milder ventricular dysfunction should, if possible, be controlled with optimum doses of digitalis and/or diuretics before ISOPTIN is used (Note interactions with digoxin under Precautions) ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild and controlled by decrease in ISOPTIN dose) Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported Such elevations may disappear even with continued treatment, howreported. Such elevations may disappear even with continued treatment, however, four cases of hepatocellular injury by verapamil have been proven by rechallenge. Periodic monitoring of liver function is prudent during verapamil therapy. Patients with atnal flutter or fibrillation and an accessory AV pathway (e.g. W-P-W or L-G-L syndromes) may develop increased antegrade conduction across the aberrant pathway bypassing the AV node, producing a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually. ventricular response after receiving ISÓPTIN (or digitalis). Treatment is usually D.C. cardioversion, which has been used safely and effectively after ISOPTIN Because of verapamil's effect on AV conduction and the SA node, 1° AV block and transient bradycardia may occur. High grade block, however, has been infrequently observed. Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high mortality rate and that most were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia. 2° AV block, sinus arrest, pulmonary edema and/or severe hypopatricular discontinuation. bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. Most adverse effects responded well to dose reduction and only rarely was verapamil discontinued. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used along if possible. If combined therapy is used along if possible if combined therapy is used along if the possible is combined therapy is used along if the possible is combined therapy is used along if the possible is combined therapy is used along if the possible is combined therapy is used along the possible is combined therapy in the possible is combined to the possible is combined the possible is combined therapy in the possible is combined the possible in the possible is combined the possible is combined the possible is combined the possible is combined to the poss be used alone, if possible. If combined therapy is used, close surveillance of vital signs and clinical status should be carried out. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of ment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patients should be carefully monitored to avoid over- or under-digitalization. ISOPTIN may have an additive effect on lowering blood pressure in patients receiving oral antihypertensive agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Clinical experience with the concomitant use of ISOPTIN and short- and long-acting intrates suggest hepeficial integration without undesirable drug integrations. Adequate anisest hepeficial integration without undesirable drug integrations. gest beneficial interaction without undesirable drug interactions. Adequate ani mal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test *Pregnancy Category C*. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in delivery only it clearly needed. It is not known whether verapamil is excreted in breast milk, therefore, nursing should be discontinued during ISOPTIN use **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block. 3rd degree (0.8%), bradycardia. HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported (See *Warnings*.) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred wision, syncope, muscle cramp, shiped: ness, claudication, hair loss, macules, spotty menstruation How Supplied: ISOPTIN (verapamil HCI) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side Revised August, 1984



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The Use of Antidepressants, Anticonvulsants and Neuroleptic Drugs in Treating Pain

WAYNE O. EVANS, Ph.D. Indianapolis

Antidepressants

In considering the use of antidepressants as adjunctive or sole drugs in the treatment of pain, one can become somewhat confused because of the association of chronic pain and depression. In every chronic illness, depression should be assessed, and, if it is found, aggressively treated. However, it has been found that, in certain patients, even if they do not have the signs of clinical depression, antidepressant drugs can be useful either by themselves or as an adjunct to other treatments.

The antidepressant drugs of themselves have not been shown to be analgesic in experimental pain models. There is some small degree of evidence that they may act to potentiate the effects of narcotics.³ The antidepressant drugs of the tricyclic types have been studied in migraine, tension headache, facial pain, postherpetic neuralgia, trigeminal neuralgia, peripheral neuropathy, low back pain, chest pain, fibrositis, arthritis, and in chronic pain patients of varying disorders. These have been reviewed by Aronoff, Evans, Lee and

Spencer. The drugs that have been most studied have been those that are of the serotonin-sparing type such as amitriptyline (Elavil) and doxepin (Adapin, Sinequan). However, both imipramine (Trofranil) and desipramine (Norpramin, Pertofrane) also have been studied. All in all, response rates from studies using antidepressant medications vary from 44 to 70% with an average of about 62% of patients finding a significant relief of pain.

The regimen of tricyclic amines in pain generally does not follow that of their use in major depressive episodes, even if the patient is depressed. It has been found that these drugs can be used in much lower doses with equal therapeutic efficacy. Indeed, in the elderly, doses as low as 10 or 25 mg in the evening have been found to be efficacious. In the average patient, doses generally are started at 50 to 75 mg in the evening; then, proceeding by upward titration, the dosage is increased to perhaps no more than 150 mg at night. The time to onset of action seems to be a little faster than that which one would expect in treating a major depressive episode. It has been this author's experience that some degree of pain relief becomes evident within the first week of the administration of the drug.

When signs of a major depressive episode are present, as is usually the case in chronic pain, one will begin to see the reduction in sleep problems associated with depression within the first few days. This, along with the general side effects of dry

mouth, urinary retention, heart palpitations, sedation and dizziness should be considered as a good sign that the drug is active at a biological level. These side effects generally become tolerated within two weeks of taking the drug.

The adverse effects to all of these drugs are their anticholineragic effects of dry mouth, blurred vision and constipation, but they are usually mild and subside as therapy is continued. Cardiovascular effects include tachycardia and hypotension. One should be careful in using these drugs in persons with heart problems. Other infrequently reported side effects are extrapyramidal symptoms, gastrointestinal reactions, secretory reactions such as increased sweating, weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash, and pruritus. In certain cases, particularly when dealing with peripheral neuralgias, if the tricyclic amine by itself does not produce the desired effect, the drug, fluphenazine (Prolixin) at 1 mg TID may be a useful addition.

Some patients will not respond effectively to the tricyclic antidepressants. For these it is possible to use monoamine oxidase inhibitors or lithium for the atypical depressions that sometimes accompany chronic pain.

A relative newcomer to the treatment of depression that is associated with chronic pain is the drug, alpra zolam (Xanax). There have been numerous documented studies showing it to be an effective antidepressant

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drug which acts within four to five days. There are no specific reports at this time which give evidence that it has any direct effect on pain, although its actions as an anxiolytic may well help decrease pain through reduction in muscle tension. In patients with agitated depressions, those who are particularly tense and tight, or patients in whom tricyclic amines are contraindicated, this seems to be a useful addition to our

Anticonvulsants

Anticonvulsive therapy has been shown to be effective with many types of neurological disorders. These include trigeminal neuralgia, glossopharyngeal neuralgia, superior laryngeal neuralgia, tabetic lightening pains, diabetic neuropathy, post herpetic neuralgia, phantom limb pain, multiple sclerosis, thalamic pain syndromes and various other neuralgias."

Four basic anticonvulsant agents have been studied for neurologic pain carbamazepine (Tegretol), clonazepam (Clonopin), phenytoin (Dilantin), valproate (Valproie Acid). Whether any one of these given drugs is more effective for one type of pain or another of neurologic origin remains in great doubt. Rather, it seems that some patients will benefit from one drug and other patients from another drug regardless of the particular type of neurologic pain that one is dealing with. For this reason, the best way to go about testing the drugs is to try each in sequential order to determine which works best for the patient. Probably the best order of administration for testing would be first, carbamazepine; seeond, clonazepam; third, phenytoin; and finally, sodium valproate.

Drugs should be administered until they come to the full therapeutic blood levels which would be for car bamazepine = 21.2 µmol L; clonaze pam = 0.064 µmol L; phenytoin=15.6 µmol L; valproate = 391 µmol L.

TABLE									
Usual	Therapeutic Drug	Doses							

	Initial	Maximum
carbamazepine	100 mg BID	400 mg BID
elonazepam	1 mg TID	6-8 mg TID
phenytoin	300 mg/day	400 mg/day
valproate	5 mg TID	20 mg TID

Patients should be left upon an effective therapeutic dose of the drug for approximately two weeks before the administered drug is reduced and the next drug tried. The *Table* shows usual drug doses.

The common adverse effects of the anticonvulsive drugs are mental slowing, sedation and mental retardation. Further, with all of these drugs, nystagmus attacks, slurred speech, mental confusion, dizziness, transient nervousness, motor twitch ings and headaches have been observed. The intestinal symptoms include nausea, vomiting and constipation. Occasionally, though rarely, one will have dermatological manifestations which can include thrombocytopenia, leukopenia, granulo cytopenia, agranulocytosis, and pancytopenia.

In patients who have compromised liver function, these drugs should be used with great care. In the use of all these drugs it is wise, particularly in the initial months of use, to follow the blood for various possible dyscrasias. This is particularly true for carbamazepine. Clonazepam, as with any of the benzodiazepine derivatives, is contraindicated in acute angle glau coma.

Neuroleptic Drugs

Neuroleptic drugs are generally used in combination with some other drug.

A particularly popular combination is promethazine (Phenergan) in combination with one of the narcotics. This is not necessarily a rational choice since there are studies which show that promethazine is actually antagonistic to analgesic action and

produces an overly sedated patient. Such combinations should be examined to determine whether it is better to administer individual drugs at the appropriate dose for the patient rather than to use fixed drug combinations. As was mentioned earlier, the neuroleptic drug fluphenazine (Prolixin) is sometimes used in association with a tricyclic amine for pain of neurologic disorders. Perphenazine (Etrafon, Triavil, Trilafon) and chlorprothixene (Taractan) have also been used in these types of treatments.

The only phenothiazine which has proven analgetic effectiveness is methotrimeprazine (Levoprome). 1111 It has been used for pain of labor, carcinoma, headache, miscellaneous injuries, rheumatoid arthritis, spinal injury, fractures, cardiac pain, peptic ulcer and other painful conditions. It generally is used in a dose range of between 10 and 20 mg. Its time action is quite long, with significant percentages of patients responding with good pain relief, for between two to four hours. It has been used in pediatric patients at a dose of 0.15 mg per ib.

This drug is only available for parenteral use in single dose ampules of 1cc containing 20 mg and in multiple dose vials of 10cc and 30cc containing 20 mg per ce. It should be diluted with isotonic saline and injected slowly or added to any other fluid infusion to avoid irritation of the veins.

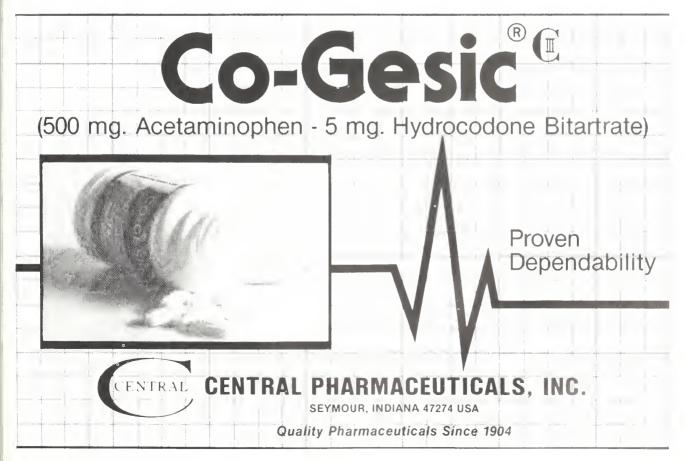
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'Asanguinous' Open-Heart Surgery

PHILIP S. CHUA, M.D. CRIS J. CARLOS, M.D. FELIX R. GOZO JR., M.D. VICTOR K. O'YEK, M.D. Merrillville

Used Routinely
as an Adjunct
to Other Blood
Conservation
Measures, the
Authors Find That
Autologous Transfusion
Is Safe, Practical,
Cost-effective and
Efficient . . .

sanguinous is a term we used in this paper to dramatize the fact that open-heart surgery could be performed safely without any homologous blood transfusion, and to underscore the feasibility, practicality and wisdom in utilizing autologous blood transfusion, as one of the prudent measures in the conservation of blood in open-heart surgery.

We find it most interesting and apropos to present this paper on autologous transfusion at this international meeting in London, particularly aware of the fact that the following two important historical events took place here in London.

The first successful transfusions in human beings were performed in 1667. Lower, before the Royal Society in London, took the blood of a sheep and transfused it to a patient who had 6 to 7 ounces of his blood removed in the hopes of altering his "unbalanced" character.

The first use of autologous transfusion was reported by Duncan in the British Medical Journal in 1886 in a patient with crushed leg from a railway injury. While amputating the leg, Duncan collected the shed blood in a dish with phosphate of soda and returned it to the patient.

It was not until the 20th century that autotransfusion was reported in the United States. In 1916, Lockwood first used this technic for splenectomy for a patient with Banti's disease, for traumatic rupture of an abdominal viscus with severe hemorrhage and for ruptured extrauterine pregnancies.

It is the purpose of this paper to share with you our modest experience with autologous transfusion in open-heart surgery in a small community hospital, covering the period January 1982 to August 1984. We initiated this auto transfusion program January 1, 1982.

There are now three surgical teams involved in this project at the St. Anthony Medical Center, a 400-bed private hospital in Crown Point, Indiana, which is about 40 miles southeast of Chicago.

Our routine includes the use of the Terumo hollow-fiber membrane oxygenator, with non-blood prime and techniques in open-heart patterned after Dr. Cooley's, with minor modifications. Since the submission of our abstract, we have included 129 more cases for this year.

Phlebotomy for predeposit of two units of blood a day before surgery, and two units immediately before going on bypass, was done in only one patient, a Jehovah's Witness who underwent mitral valve replacement in January of 1982.

The protocol we use in our autotransfusion program in open-heart surgery is as follows:

1) Any medication that impairs coagulation is discontinued at least seven days prior to surgery;

2) Five units of homologous packed cells and five units of fresh frozen plasma are stored in the blood bank for possible use. For valve cases, we also order 10 units of platelets on hold;

3) Meticulous surgical hemostasis is observed in the operating room.

After cessation of cardiopulmonary bypass, and after protamine has been administered, blood in the pericardial sac, and in the oxygenator, is gently aspirated into Sorensen collecting bags, using negative suction of not more than 100mm. The tubing connected to the suction has a small sidelumen through which heparin solution (25,000 units in 500cc NS)—in

Presented at the Fourth International Scientific Symposium of the Denton A. Cooley Cardiovascular Surgical Society, Sept. 17, 1984, London.

Correspondence: Cardiovascular Surgery Associates, 8684 Connecticut St., Merrillville, Ind. 46410. continuous drip—is mixed with the blood that is aspirated from the pericardial sac, or the pleural cavity, if it is open.

The Sorensen bag is delivered to the blood bank for cell washing. Our laboratory uses the IBM Cell Washer Model No. 2991, 1 & 2. The turnaround time is about 22 minutes.

This autologous blood is transfused to the patient who, by this time, is in the Cardiovascular Intensive Care Unit.

If the hematocrit falls below 20 while on CPB, or below 27% post-CPB, homologous blood is added to the pump prime.

During the ensuing hospitalization days prior to discharge, homologous blood is transfused if the hematocrit is below 30%, especially in the elderly.

This series includes 458 patients in 1982, 528 in 1983, and 303 from January to August of 1984, with a total of 1,289 patients. 748 were males and 541 were females. The age range for males was 35 to 72 years of age; for females, 26 to 68 years of age.

Coronary bypass was performed on 1,121 patients, 32 with concomitant repair of LV aneurysm, and 27 with unilateral carotid endarterectomy.

168 underwent cardiac valve replacement, 109 mitral, 57 aortic, and two combined MVR and AVR. Three of the MVR, and five of the AVR had concomitant CABG.

OPERATIVE MORTALITY:

CABG									1.2%	(14)
AVR.									3.5%	(2)
MVR.									2.7%	(3)

Total No. of Specimens Received
•
by Blood Bank 574
Total Volume Salvaged Fluid
Received 849,878 ml.
Total Volume of Washed Cells
Returned 146,343 ml.
Total Equivalent RBC Value per
225 ml 650 units
In 1982 an average of 55 units of
packed cells per month were sal-

Total Volume of Salvaged Fluid Received 1,276,092 ml.

Total Volume of Washed Blood Returned...... 248,350 ml. Total Equivalent RBC Value per

In 1983 the average blood salvaged was 92.1 units per month.

From January to August 1984, this technic of blood salvage yielded and saved 645 units of washed red cells for an average of 80 units per month.

All patients in this series received autologous transfusion. Only 180 patients, or 14%, received homologous blood. 1,109 patients, or 86%, did not require homologous transfusion.

The hematocrit count on the IBM washed blood was 82-88%.

On cardiopulmonary bypass, hematocrit was maintained at 20-22%.

Two hours post-bypass, the count was 27-28%.

After an average of 1.8 units of autologous transfusion, the hematocrit went up to 34-36% in 86% of cases.

135 of the 180 who received homologous blood transfusion were valve replacements (about 80%).

There were no coagulopathies, air embolism or other adverse reactions or complications noted in this series.

By the time the patients were discharged, usually between seven to 10 days post-op, the hematocrit count among those who received only autologous blood ranged between 36-38%.

In summary, autologous transfusion, used routinely as an adjunct to other prudent blood conservation measures in open-heart surgery, is safe, cost-effective, practical and efficient.

Where indicated, homologous transfusion of blood and blood components should be judiciously utilized.

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Skin Diseases: Current Concepts, Therapy

2. PSORIASIS

BRIAN POTTER, M.D. Michigan City

Page 14 Soriasis is a common, often familial disease of the skin, the cause of which is unknown. Increased predisposition to the disease is associated with the inheritance of specific HLA antigens, namely HLA-B13, B17, B37, and most strongly with HLA-Cw6.

Acute psoriasis can be precipitated by streptococcal throat infections, and presents as fairly generalized, small, superficial, pink lesions. Conversely, chronic psoriatic lesions tend to be distributed on the skin over pressure points, particularly the scalp, elbows and knees, and in superficial wounds where the lesions take the same shape as the injury. The lesions are erythematous, papular and coalescent in defined plaques with a silvery scaling.

Psoriasis is a disease with characteristic changes in both epidermis and dermis. The specific dermal abnormality is in the papillary capillaries, which become clongated, convoluted, tortuous, dilated, and proliferative. This is associated with the enlargement and superficial proliferation of the dermal papillae, and with changed

characteristics of the capillary loop from arterial type to venous, with increase of blood flow to twice the normal, the development of gaps between endothelial cells, and an exudate of inflammatory cells and serum.

Epidermal hyperplasia occurs with the vascular proliferation and, in fact, the initiating factors in psoriasis appear to be epidermal.2 The normal differentiation that occurs within the epidermis is reduced. The two most prominent morphological changes in the epidermis are the absence of developed keratohyalin granules, and the persistence of remnants of keratinocyte nuclei into the corneal layer. The glycoprotein surface layer that basal cells normally acquire as they ascend toward the corneal layer is diminished or lacking in psoriasis. Neutrophilic leucocytes tend to migrate into the epidermis and accumulate in small pockets within the epidermis and horny layer.

The entire skin of psoriatic patients is abnormal. This is the reason why physical stimuli cause lesions to appear, and why certain medications precipitate psoriasis in predisposed persons, including chloroquin, lithium, antihypertensive beta-blocking drugs, and the withdrawal of corticosteroids.

Rapid proliferation of epidermal cells in psoriasis is associated with increased anabolic activity. The population of germinative epidermal cells engaged in the synthesis of DNA is increased, and the duration of this process is decreased. A basic defect exists in the control of nucleic acid synthesis, DNA and RNA being incompletely hydrolysed. Uric acid, the

end product of purine metabolism, is increased in the blood level of most psoriatics. The germinative cycle is more rapid, and the time taken by cells passing through the epidermis is shorter than normal. All this results in the characteristic scaling of psoriasis.

Increased amounts of epidermal glycogen, four to five times the normal, are found in psoriatic lesions, and greater amounts of the enzymes of carbohydrate metabolism. Lipid metabolism and the cholesterol/ester ratio also increase.

The psoriatic lesion thus represents the aggregate of an increased number of epidermal cells, rapid rate of cell reproduction, and excessive production of the end product, namely, stratum corneum. Inflammation is not marked except in psoriatic erythroderma, generalized pustular psoriasis, and psoriatic arthritis, in which the synovium is involved as well as the skin.

The principle and purpose of therapy in psoriasis is to restore to normal the accelerated growth rate of the epidermis. The methods available for this include emollient ointments, sometimes under occlusion, corticosteroids, anthralin, coal tar, systemic methotrexate, ultraviolet light, and combinations of these. Systemic steroids are contraindicated in psoriasis, except perhaps in acute guttate psoriasis, because of their consequent suppression of the hypothalamic-pituitary-adrenal axis. The danger of generalized pustular psoriasis tends to complicate the withdrawal of steroids, and even to break through during the treatment.

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Topical corticosteroids. Although psoriasis responds effectively to top ical therapy with the most potent corticosteroids, for instance halcinonide, amcinonide, fluocinonide and others, they cannot be recommended for prolonged use in all forms of this chronic disease. This is because of their atrophying effects, the rapid development of tolerance to topically applied corticosteroids, the possibility of secondary Cushing's syndrome during their use, and pustular psoriasis on their withdrawal. Corticosteroid applications in psoriasis should therefore be limited to short term use, for the scalp, intertriginous regions, and inflamed lesions intolerant to other topical antipsoriatic preparations.

Anthralin (dithranol, eignolin, trihydroxyanthracene) is a compound that alters the metabolism of cells and decreases their mitotic rate. It is therefore useful in the reduction of thick, chronic, psoriatic plaques. Anthralin is available as 0.1 to 1% cream (Lasan, Drithoereme) and ointment (Anthra-Derm). The disadvantages of anthralin include conjunctivitis if it inadvertently reaches the eye, irritation of the normal skin surrounding the lesions, and staining of linen. These can to some extent be avoided by removal of the preparation after a short time. A beneficial effect is obtained even from a one-hour application of 1% anthralin, which is equivalent to 0.1-0.4% applied for 8-12 hours.

Tars are mixtures of indefinite numbers of aromatic hydrocarbons including naphthalene, cresol. phenols, sulfur compounds and black pitch. The phenols are anesthetic and induce reformation of the normal granular layer in parakeratotic, diseased epidermis. The neutral and basic fractions lead to hypertrophy of epidermal cells, and increased thickness of the epidermis. Pitch absorbs incident light and is photosensitizing.

Most of these effects are useful in the treatment of psoriasis.

Coal tar, modified for application, is supplied commercially in hydroal-coholic gels, as Estar (5% concentration) and Psorigel (7.5%), 30% emulsion (Zetar), and alcoholic solution, USP. One ounce of either of the last two can be added to the bathwater for general application. For the scalp they can be compounded in 10% concentration in Nivea oil or tincture of green soap, rubbed in and left on under a shower cap overnight and shampooed out in the morning.

Disadvantages of coal tar include messiness and the induction of folliculitis. Although tar is carcinogenic in experimental animals, no increase in carcinogenicity has been documented in humans in over 50 years of use.

Coal tar is photosensitizing, as already mentioned. The combination of applications of tar and exposure to ultraviolet light is effective and is known as the Goeckerman treatment. The tar is allowed to remain on the skin for 2-12 hours, then removed with mineral oil before ultraviolet irradiation, regular sunlamps being used."

Ultraviolet light. In fact, ultraviolet alone has a beneficial action. The only preparation required on the skin is to soak the lesions with water-miscible ointment or mineral oil, for better penetration of the light through translucent scales. The treatment requires erythema-producing dosage, the dose received being proportional directly to the duration of exposure and inversely to the square of the distance from the source.

The mechanism of the effect of ultraviolet light on psoriatic lesions is unknown. However, the epidermis becomes more uniform in thickness and the granular and corneal layers reform. Cytotoxic effects produced in the epidermis, which are beneficial in psoriasis, include reduction in mitotic rate, prolongation of cell turn-

over time, suppression of DNA synthesis, inhibition of RNA and synthesis of protein, and decreased en zymatic activity.

Sunlight is naturally filtered through the atmosphere, which prevents wavelengths shorter than 290 nanometers from reaching the earth's surface. The ultraviolet spectrum that reaches the earth comprises, there fore, wavelengths of 290-400 nm, light of longer wavelength being visible. This spectrum is artificially and conceptually divided at 320nm into short wave UVB and long wave UVA. UVB, 290-320 nm, can be filtered out by glass, including ordinary types of window glass. This part of the spectrum is the most effective in causing sunburn, but all ultraviolet, in sufficient dosage, causes ervthema, and improves psoriasis. The shorter wavelengths are more efficient because their photons are of higher energy and, when absorbed by tissue, are most active in producing photochemical changes and altering cell metabolism.

The sources of UVB include sunlight, high-pressure mercury are lamps, and batteries of fluorescent sunlamps, such as Westinghouse FS20 or FS40. Exposure should begin at less than the erythema-producing dose, and be increased at each subsequent treatment until the skin reacts with erythema. The course may then be continued at this dose, at daily intervals, until the skin is clear of psoriasis or shows only pink or hyperpigmented remains of lesions. Remission usually takes two to four weeks.

UVA is produced by fluorescent black light lamps such as the GE black-light or BLB F72T12/BL/HO. Treatment with UVA alone would require such high intensity and long duration of exposure that it is impractical without the concomitant use of photosensitizers, which greatly reduce the amount of long wave ultraviolet radiation required. Photoactive furocoumarins are the photosensitiz-

ers used with UVA in the PUVA technique. This requires special apparatus, however.

Antimetabolite treatment, Psoriasis responds well to methotrexate but, because of its toxicity, this drug is advised only in severe cases with discomfort and incapacity unresponsive to other therapy. Methotrexate is an analog and antagonist of folie acid, given orally, intramuscularly or intravenously. The drug acts by binding the enzyme dihydrofolate reductase, thus inhibiting the conversion of folic acid to tetrahydrofolic acid. This is a coenzyme involved in the biosynthesis of thymidylic acid and the purine ring of nucleic acids. Methotrexate inhibits replication of epidermal and other cells by means of this interference with the synthesis of DNA.

Methotrexate is not metabolized but is excreted unchanged in the urine. It induces nonspecific hepatotoxicity, but particularly portal fibrosis. Patients who drink alcohol develop abnormal findings in liver biopsies more frequently than others who do not. Changes in the liver are fewer when methotrexate is given on an interrupted schedule than when it is given daily. The dose is 2.5 to 7.5 mg of methotrexate orally at 12-hour intervals for three doses only, each week. The maximum dose is 30mg weekly, which leads to clinical improvement of psoriasis in most patients.

Deaths of patients after methotrexate therapy have been from intramuscular οr intravenous administration rather than oral, and have been due to hepatotoxicity, gastrointestinal hemorrhage, leukopenia, renal failure, thromboembolic disease, cardiac insufficiency, and secondary infection. Other adverse reactions have included thrombocytopenia, macrocytic anemia, mucosal ulceration, nausea, vomiting, fever, chills, headache, oligospermia and teratogenicity.

Methotrexate is therefore contraindicated in pregnancy, hepatitis, fibrosis or cirrhosis of the liver, peptic ulceration, abnormal renal function, infectious disease, and alcoholism. All female patients must avoid pregnancy. Liver biopsy should be done before methotrexate therapy is begun, as well as blood counts which must be repeated weekly before giving the next dose, and renal and liver function studies which should be repeated within three to four months.⁹

Hydroxyurea (Hydrea) is another antimetabolite useful in the treatment of psoriasis, the dose being 500mg two or three times daily in proportion to the weight. This drug blocks the production of thymidine and its incorporation into DNA. It is active only during the DNA synthesizing phase of the cell cycle.

Peak concentration in serum is reached about two hours after ingestion, and the drug is no longer detectable in the blood after 24 hours, 80% being excreted unchanged in the urine. It is therefore contraindicated in the presence of renal failure and, since it is teratogenic, in pregnancy. Adverse reactions have included leukopenia, thrombocytopenia, macrocytic anemia, nausea, vomiting, diarrhea, fever, malaise, myalgia, mucosal ulcerations and alopecia.

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INDICATIONS AND USAGE: These preparations are indicated for the treatment of infections caused by susceptible strains of designated microorganisms as follows: Respiratory Tract Infections (e.g., tonsillitis, pharyngitis, and lobar pneumonia) due to *S. pneumoniae* (tormerly *D. pneumoniae*) and group A beta-hemolytic streptococci [penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever; Velosef (Cephradine, Squibb) is generally effective in the eradication of streptococci from the nasopharynx; substantial data establishing the efficacy of Velosef in the subsequent prevention of rheumatic fever are not available at present], Otitis Media due to group A beta-hemolytic streptococci, *H. influenzae*, staphylococci, and *S. pneumoniae*; Skin and Skin Structures Infections due to staphylococci and beta-hemolytic streptococci; Urinary Tract Infections, including prostatitis, due to *E. coli*, *P. mirabilis*, *Klebsiella* species, and enterococci (*S. faecalis*).

Note: Culture and susceptibility tests should be initiated prior to and during therapy.

CONTRAINDICATIONS: In patients with known hypersensitivity to the cephalosporin group of antibiotics.

WARNINGS: Use cephalosporin derivatives with great caution in penicillinsensitive patients since there is clinical and laboratory evidence of partial cross-allergenicity of the two groups of antibiotics; there are instances of reactions to both drug classes (including anaphylaxis after parenteral use). In persons who have demonstrated some form of allergy, particularly to drugs, use antibiotics, including cephradine, cautiously and only when absolutely necessary.

Pseudomembranous colitis has been reported with the use of cephalosporins (and other broad spectrum antibiotics); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with antibiotic use. Treatment with broad spec-

trum antibiotics alters normal flora of the colon and may permit overgrowth of clostridia. Studies indicate a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis. Cholestyramine and colestipol resins have been shown to bind the toxin *in vitro*. Mild cases of colitis may respond to drug discontinuance alone. Manage moderate to severe cases with fluid, electrolyte and protein supplementation as indicated. Oral vancomycin is the treatment of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile* when the colitis is severe or is not relieved by drug discontinuance; consider other causes of colitis.

PRECAUTIONS: General: Follow patients carefully to detect any side effects or unusual manifestations of drug idiosyncrasy. If a hypersensitivity reaction occurs, discontinue the drug and treat the patient with the usual agents, e.g., pressor amines, antihistamines, or corticosteroids. Administer cephradine with caution in the presence of markedly impaired renal function. In patients with known or suspected renal impairment, make careful clinical observation and appropriate laboratory studies prior to and during therapy as cephradine accumulates in the serum and tissues. See package insert for information on treatment of patients with impaired renal function. Prescribe cephradine with caution in individuals with a history of gastrointestinal disease, particularly colitis. Prolonged use of antibiotics may promote the overgrowth of nonsusceptible organisms. Take appropriate measures should superinfection occur during therapy. Indicated surgical procedures should be performed in conjunction with antibiotic therapy.

Information for Patients: Caution diabetic patients that false results may occur with urine glucose tests (see PRECAUTIONS, Drug/Laboratory Test Interactions). Advise the patient to comply with the full course of therapy even if he begins to feel better and to take a missed dose as soon as possible Tell the patient he may take this medication with tood or milk since G.l. upset may be a factor in compliance with the dosage regimen. The patient should report current use of any medicines and should be cautioned not to take other medications unless the physician knows and approves of their use (see PRECAUTIONS, Drug Interactions).

Laboratory Tests: In patients with known or suspected renal impairment, it is advisable to monitor renal function.

Drug Interactions: When administered concurrently, the following drugs may interact with cephalosporins:

Other antibacterial agents — Bacteriostats may interfere with the bactericidal action of cephalosporins in acute intection; other agents, e.g., aminoglycosides, colistin, polymyxins, vancomycin, may increase the possibility of nephrotoxicity

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Carcinogenesis, Mutagenesis: Long-term studies in animals have not been performed to evaluate carcinogenic potential or mutagenesis.

Pregnancy Category B: Reproduction studies have been performed in mice and rats at doses up to 4 times the maximum indicated human dose and have revealed no evidence of impaired fertility or harm to the fetus due to cephradine. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, use this drug during pregnancy only if clearly needed.

Nursing Mothers: Since cephradine is excreted in breast milk during lactation, exercise caution when administering cephradine to a nursing woman.

Pediatric Use: Adequate information is unavailable on the efficacy of b.i.d. regimens in children under nine months of age.

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DOSAGE: Adults — For respiratory tract infections (other than lobar pneumonia) and skin and skin structure infections: 250 mg q 6 h or 500 mg q. 12 h. For lobar pneumonia: 500 mg q. 6 h or 1 g q. 12 h. For uncomplicated urinary tract infections: 500 mg q. 12 h; for more serious UTI, including prostatitis, 500 mg q. 6 h or 1 g q. 12 h. Severe or chronic infections may require larger doses (up to 1 g q. 6 h). For dosage recommendations in patients with impaired renal function, consult package insert.

Children over 9 months of age — 25 to 50 mg/kg/day in equally divided doses q. 6 or 12 h. For otitis media due to *H. influenzae*: 75 to 100 mg/kg/day in equally divided doses q. 6 or 12 h but not to exceed 4 g/day. Dosage for children should not exceed dosage recommended for adults. There are no adequate data available on efficacy of b.i.d. regimens in children under 9 months of age.

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Clinico-Pathologic Conference: 60-Year-Old Man with Weakness and Respiratory Failure

Edited by DOUG REX, M.D. Indianapolis

Grand Rounds—
Indiana University
School of Medicine

Dr. Rex is Chief Resident in Medicine, Indiana University Hospital W-587, 926 W. Michigan St., Indianapolis, Ind. 46223.

This is an edited transcript of the clinico pathologic conference conducted Feb. 6, 1985 during a Grand Rounds session of the Dept. of Medicine, Indiana University School of Medicine.

Discussants:

John Kincaid, M.D., Dept. of Neurology; Vernon Vix, M.D., Dept. of Radiology; Saeed T. Vakili, M.D., Dept. of Pathology.

Dr. Kincaid is Assistant Professor of Neurology and Director of the Electromyography Labs at the Indiana University Medical Center. He has established a fellowship in EMG at I.U. and has active research interests in the refinement of electro-physiologic testing for the diagnosis of neuromuscular disease and in computer-assisted EMG.

60-YEAR-OLD white male was ad mitted to another hospital. He had been well until three weeks previously when he noted the onset of neck weakness followed by upper extremity weakness and dysphagia. Shortly after hospitalization his symptoms progressed and culminated in respiratory failure requiring mechanical ventilation and tracheostomy. Episodic hypotension accompanied by bradycardia required temporary insertion of a demand pacemaker, A cystometrogram demonstrated inability to generate a voiding contraction and a Foley catheter was inserted. Lumbar puncture four weeks after the onset of weak ness demonstrated a cerebrospinal fluid (CSF) protein of 233 mg% and glucose of 56 mg% with 29 WBC/ mm³, mostly lymphocytes with a few mononuclear cells. Stains and cultures of CSF for bacteria, myeobacteria and fungi were negative. A left lower lobe pneumonia responded to antibiotics.

Ten weeks after the onset of the illness the patient was transferred to the Medical Center with the diagnosis of Guillain-Barré syndrome with respiratory failure. He denied having pharyngitis, upper respiratory tract infection, immunizations or use of any medications in the several months prior to the illness. He seldom drank alcohol but had a 60 pack-year smoking history. There was no family history of neurologic syndromes. He was an office worker and had no known exposure to toxic chemicals.

Examination revealed the blood pressure was 90/74 mm Hg, pulse 70,

temperature 37° There were no spontaneous respirations while being mechanically ventilated at a rate of 10. A tracheostomy tube was in place. He was alert and fully oriented. Rales were noted over the left postero-basilar lung field. There was no palpable lymphadenopathy and no hepatosple nomegaly.

Neurologic examination demonstrated neck weakness in all directions, especially rotation and flexion. Tests of cranial nerves other than XI were normal. Muscle tone was decreased in all limbs. Strength in the proximal muscles of the upper and lower extremities was limited to some movement against gravity. Distal musele strength was better but still the patient had easily detectable weakness, except the right wrist dorsiflexors, where strength was limited to movement against gravity. The muscle stretch reflexes were diffusely diminished, being absent at the ankles and 1+ elsewhere. There was a Babinski on the left and an equivocal Babinski on the right, Position and vibration sense were normal. Pain sensation was diminished distally in all extremities but normal with proximal stimuli. Cerebellar function and gait were untestable.

The WBC was 10,600, Hgb 12.6 gm%, the Na $^+$ 137 mEq/L, the K $^+$ 3.0 mEq/L, the C1 $^-$ 100 mEq/L and the HCO, $^-$ 28 mEq/L. The blood urea nitrogen was 8 mg/100 ml, the Cr 0.7 mg/100 ml, the glucose 90 mg/100 ml, the calcium 8.8 mg/100 ml and the magnesium 1.6 mEq/L. The serum glutamic oxaloacetic transaminase was 35 IU/L, the alkaline phospha-

tase 94 IU/L and the bilirubin 0.8 $\,$ mg/100 ml.

A chest x-ray demonstrated a left lower lobe infiltrate. The electrocardiogram was normal.

The Westergren sedimentation rate was 64 mm/hr. Tests for anti-nuclear antibody and rheumatoid factor were negative. The serum thyroxine was 10.8 ng/100 ml and the free thyroxine index 9.6 ng/100 ml. A 24-hour urine collection for heavy metals was normal. A urine screen for porphobilinogen and delta-amino levulinic acid was negative. Lumbar puncture 11 weeks after the onset of illness demonstrated CSF protein of 84 mg% and glucose 59 mg%. There were 9 WBC/mm³, all lymphocytes. CSF VDRL, cryptococcal Ag and all stains and cultures were negative. CSF electrophoretic studies were interpreted as consistent with a systemic inflammatory process, early manifestations of a lymphoproliferative disorder, or a neoplasia of the GI or respiratory tract. An electromyelogram (EMG) and nerve conduction velocity were consistent with diffuse polyneuropathy with moderate involvement of the upper and severe involvement of the lower extremities. Neurologic consultants agreed that the diagnosis was that of atvpical Guillian-Barré syndrome.

The forced vital capacity was 1.2 liters. Sputum and urine cultures grew *Enterobacter aerogenes* and appropriate antibiotics were given.

The patient had only transient neurologic recovery and had a long and difficult hospital course. Left lower lobe atelectasis developed and the left hemidiaphragm elevated. Fluoroscopy revealed only minimal but not paradoxical motion of the left diaphragm. Phrenic nerve terminal latencies were normal. A left pleural effusion developed and demonstrated 3060 RBC/mm³ and 430 WBC/mm³ with 32% polys, 59% lymphs and 9% eosinophils. The serum/pleural fluid ratios of protein and LDH were .6 and .5, respectively. Cultures and cy-



FIGURE 1: A chest radiograph from early in the hospital course demonstrates left lower lobe atelectasis.

tology were negative.

A transient improvement in vital capacity allowed 4 weeks without mechanical ventilation but infection led again to ventilator dependence. Five separate bronchoscopies demonstrated mucous plugging and edema in left lower and/or upper lobe bronchi. Cultures grew various gram negative bacteria and numerous washings and brushings for cytology specimens were negative.

Serial neurologic examinations and EMGs were essentially unchanged. A Tensilon test was negative. An eightweek course of steroids was given without apparent benefit.

In the eleventh hospital month a focal motor seizure disorder affecting the right side of the body developed. Metabolic evaluation, lumbar puncture and two CT scans of the head 10 days apart failed to reveal a specific etiology. An electroencephalogram revealed a left frontal lobe sharp wave focus. The diagnosis remained atypical Guillian-Barré.

Recurrent left lung pneumonias continued and he died 13 months after

the onset of his illness. An autopsy was performed.

Dr. Kincaíd: Dr. Vix, could we review the x-rays?

Dr. Vix: A chest x-ray from early in the hospital course (Figure 1) reveals a retrocardiac density and elevation of the left hemidiaphragm consistent with left lower lobe atelectasis. Serial films show development and resolution of several left-sided infiltrates consistent with the history of repeated pneumonias. As noted in the history, a fluoroscopic examination of the left diaphragm revealed appropriate but markedly diminished motion. An esophgram demonstrated tertiary waves but was otherwise normal.

Dr. Kincaíd: In summary, we have a previously healthy adult male who acquired a profound degree of weakness over a three-four week períod without any history of a clear-cut precipitating factor or illness. He had signs of autonomic involvement, including hypotension, bradycardia and loss of bladder function. The exam showed primarily a lower motor neuron-type weakness but there were also signs of upper motor neuron involvement, as indicated by the extensor toe sign on the left and an equivocal toe sign on the right. Laboratory findings showed an increased CSF protein and white blood cells. There was generalized peripheral neuropathy on electrical studies. He had a prolonged course of 13 months without having any significant neurologic recovery. In addition, there is a history of recurrent left-sided pneumonias which raises the question of an underlying lung disease, specifically a cancer. Finally, he developed a seizure disorder, again without any clear cause. Neurologic consultants felt he had an atypical Guillian-Barré syndrome and I would agree with that.

I shall discuss the case from the standpoint of the differential diagnosis of an acquired weakness which could develop over this time period, namely three-four weeks, and which could be this severe, that is to the point of ventilator dependence. I'll use a systematic approach in which we'll consider diseases primarily of the central nervous system (CNS), then of the anterior horn cells, the peripheral nerves, the neuromuscular junction and, finally, of muscle itself.

Any CNS disorder interrupting the descending motor tracts could produce a syndrome of this type. Generally, however, such a process will define itself by some local manifestation along the longitudinal neuraxis. Thus, in addition to weakness, we might see a brain stem sign, or perhaps a level of sensory loss. No localizing signs of this type were present in this patient. Examples of CNS disease which cause weakness include cerebral, brain stem or spinal cord infarction, intrinsic or extrinsic neoplasms of the CNS and inflammatory lesions such as transverse

myelitis. Infarctions generally manifest themselves within 12-24 hours and myelitis produces its maximum deficit within 48 hours after onset. These intervals are quite different from the three-four weeks to develop maximum deficit seen in this case. CNS neoplasms may manifest this slowly but generally are progressive lesions, whereas our patient had relatively stable neurologic deficits after reaching his maximum deficit. Additional factors against a primary CNS process include the hyporeflexia and the EMG evidence of decreased pe ripheral nerve conduction velocities (NCVs). CNS lesions which affect upper motor neurons will cause hyperreflexia and, unless there is combined lower motor neuron disease, should not affect peripheral nerve conduction. Thus, there is little to support a primary CNS process.

Diseases of the anterior horn cells typically produce weakness which may be profound. Amyotrophic lateral sclerosis (ALS) may produce the findings seen in this case but not over such a short time period. ALS develops with a subacute to chronic course in which progressive weakness appears over months to years.

When discussing the anterior horn cell we must consider polio,2 which is now a rare disease. Patients with polio generally report exposure to an infected patient or have been recently vaccinated with live vaccine. The illness begins with several days of malaise and fever followed in twothree days to one week later by the onset of paralysis. The paralysis of polio is usually asymmetric so that one arm or one leg will be predomi nantly involved and with lesser or even no involvement of other limbs. It is certainly possible, though, for the illness to progress to a complete quadriparesis including respiratory failure. The patients may occasionally have bladder involvement tran siently, although this patient seemed to have had a much longer problem with bladder function. There should be no sensory symptoms. There can be some impairment of higher cortical function and also autonomic changes probably due to involvement of brain stem nuclei by the virus. We don't have much to support the diagnosis of polio in this case, but it should be considered any time we are dealing with a rapidly progressive, profound weakness.

Another type of anterior horn cell disease we should consider is that occurring as a remote effect of a carcinoma. This has been ealled en cephalomyelitis, neuromyopathy and multiple other names. The weakness from anterior horn cell involvement in these cases is usually a part of a more general neurologic process including cerebellar defects and often profound sensory loss. The mechanism for the neurotoxicity is not clear. Autopsy reveals infiltration of the anterior horn cells and sensory ganglia by lymphocytic cells. The carcinomus are most frequently lung tumors, usually of the oat cell type, but the syndromes can be seen in breast and uterine cancer and sometimes lymphoma. My objection to this as a primary diagnosis in our case is the time course of progression. Again, our patient had reached maximum deficit by about three four weeks. Patients with anterior horn cell disease as a remote effect of carcinoma usually have a more slowly evolving process over months and even perhaps a year.

Next I'll consider peripheral nerve disorders, beginning with the Landry-Guillian-Barré syndrome (GBS). GBS is an autoimmune disorder in which the target cell is the Schwann cell. On biopsy there is a lymphocytic and monocytic infiltrate in the peripheral nerves which leads to demyelination, loss of nerve conduction and resulting weakness. Two-thirds of patients report an episode of upper respiratory tract infection or gastroenteritis one-three weeks prior to the onset of weakness. Other precipitating factors include surgical pro-

cedures and vaccination. The latter was particularly implicated after the swine flu vaccines in 1976 but has not been a problem with influenza vaccines since that time. GBS has also been described as a remote effect of neoplasms, especially Hodgkin's disease and lung cancer.

GBS patients, like the patient in our case, are afebrile at presentation and manifest primarily weakness. The weakness usually progresses in ascending fashion, and may eventually involve the cranial nerves. Occasionally, a descending paralysis is seen. Fifty per cent of GBS patients show their maximum evolution of weakness by two weeks after onset and 90% have peaked by four weeks. Sensory loss tends to be minor. Autonomic findings such as tachyeardia, bradycardia and orthostatic hypoten. sion occur in 50% of cases. Sphincter involvement, especially the urinary tract as occurred in this ease, occurs in less than 10% of cases and easts doubt on the diagnosis.

In 80-90% of eases, the CSF in GBS demonstrates increased protein by the end of the first week. This results from disruption of the blood nerve barrier by the autoimmune process. Typically in GBS there are less than 10 WBC/mm in the CSF but less than 50 is compatible with the diagnosis. Our patient had 29 WBC mm at the initial contact. The electromyogram in 60-70% of GBS cases shows a profound reduction of peripheral NCVs from a normal of 50 m sec to the 20-25 m/sec range. This patient's NCVs were about 40 m/sec, which again are atypical for, but not incompatible with, GBS.

Unlike our patient, who was ill for 13 months with little or no signs of recovery, 80% of GBS patients make a complete recovery in four six months. If the autoimmune process destroys not only the Schwann cells but also the nerve axons, then recovery may be delayed for up to several years while the nerves regenerate. Rarely no recovery at all occurs.

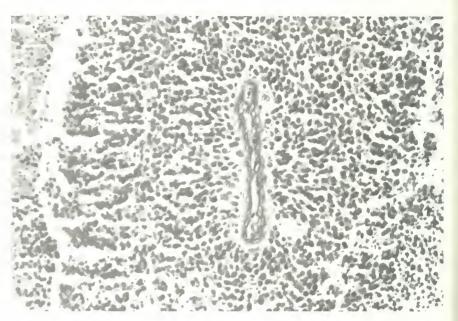


FIGURE 2: Histologic section of left lung tumor. The small cells with scant eytoplasm are consistent with out cell carcinoma (400x).

Overall there are factors which favor GBS, but this patient's syndrome is clearly atypical.

Porphyria, a genetic disorder characterized by intermittent excess heme production, must be considered in cases of peripheral neuropathy. The type most commonly associated with neuropathy is acute intermittent porphyria, in which patients classically present with a bout of abdominal pain, psychiatrie disturbances and neurologic findings. These attacks are interspersed with asymptomatic periods. Abdominal pain appears first, is acute and severe and may lead to laparotomy. Psychiatric disturbances are typically anxiety, restlessness, and insomnia. The neuropathy is the most dangerous component and may progress to quadraplegia in a few days. Cranial nerve involvement as well as central nervous system signs of obtundation and coma may be seen. The pathogenesis is presumed to be a direct neurotoxic effect of accumulated intermediates of heme synthesis. The mortality with severe neuropathy is 40% even with optimal treatment and supportive care. Phy

sicians must recall that sedatives, particularly barbiturates, may further stimulate heme production and may be fatal. The diagnosis is made by measurement of urine delta-aminolevulinic acid and porphobilinogen, which were negative in this case.

Diphtheria should also be considered in such a case. About 20% of patients with clinical infection with diphtheria develop neuropathy. The cranial nerves are involved first, and usually with dysphagia and fluid regurgitation from both nerve involvement and the severe exudative pharvngitis. The neuropathy spreads to the limbs with gradual development of paresis. Demyelination caused by the direct effect of diphtheria toxin on the Schwann cells leads to profound impairment of nerve conduction similar to that in GBS. Widespread vaccination has now made the disease rare and there is no history of fever or pharyngitis to support the diagnosis in this case.

A toxin which could produce such profound weakness with both peripheral and central signs is orthocresylphosphate. The chemical is used by



FIGURE 3: Histologic section of dorsal root ganglion demonstrates drop-out and degeneration of ganglion cells and a chronic inflammatory infiltrate (200x).

plastic manufacturers and in high temperature lubricants such as jet engine oil. Exposure is work-related and is rare in the United States and we have no history to support this diagnosis.

Several disorders of the neuromuscular junction may cause severe weakness. This patient's course is atypical for myasthenia gravis, since there are usually premonitory symptoms before profound weakness develops. Occasionally, a myasthenic will be only mildly symptomatic and undiagnosed only to have a crisis precipitated by administration of a neuromuscular blocking agent during anesthesia or an aminoglycoside antibiotic. Typically, cranial nerve signs occur first, especially ptosis and diplopia. Tensilon (edrophonium) testing should be positive and the serum contains anti-acetylcholine receptor antibodies. The Eaton-Lambert syndrome is a paraneoplastic syndrome which clinically resembles myasthenia gravis. Myasthenia gravis and the Eaton-Lambert syndrome are each associated with distinct EMG abnormalities, neither of which were

present in this case.

Ingestion of inadequately prepared food contaminated with Clost radium botulinum may result in paralysis. Toxin produced by the bacteria blocks the release of acetyl choline from the presynaptic nerve endings. Most eases in the U.S. are reported in Colorado and Utah, possibly because the high altitude causes water to boil at lower temperature which may not kill the bacterial spores. The clinical syndrome is distinct. Patients are afebrile but may have nausea, vomiting and abdominal pain. Eighteen to 36 hours after ingestion of contaminated food diplopia, dry mouth and dysphagia develop. Weakness develops in descending fashion and may cause cranial nerve and somatic paralysis. Since both nicotinic and muscarinic receptors are affected, patients classically lose their pupillary light responses, a finding not seen in this case. Sensation remains intact and the CSF is normal. With early treatment and vigorous supportive care, neurologic function is eventually recovered, unlike our patient.

The normal CPK and the EMG evi-

dence of peripheral neuropathy make either primary or paraneoplastic myositis very unlikely as the cause of this patient's weakness. Marked disturbances of potassium may also cause severe muscle weakness, but were not present in this case.

In summary, I agree with the neurologic consultants that the best diagnosis is atypical GBS. I am particularly swayed by the time course of onset and the stability of the neurologic deficits after reaching maximum. I am uncomfortable, however, with the extensor toe signs and the bladder involvement. I cannot explain the seizure disorder. There was an EEG focus but no abnormality on CT scan. The long smoking history and the recurrent lung infections raise the question of an underlying lung cancer, and I cannot rule out the possibility that this atypical neurologic syndrome represents a remote effect of a carcinoma.

Dr. Kineaid's diagnosis: atypical Guillian-Barré syndrome.

Dr. Saeem T. Vakili:

On sectioning the lungs there was a small mass in the left hilum. Histologic examination (Figure 2) showed many small cells with scant cytoplasm consistent with small or oat cell carcinoma of the lung. There were metastases to hilar lymph nodes but no distant metastases.

Gross examination of the brain was normal. Microscopic examination showed remote ischemic changes in the left hippocampus and cerebellar cortex. The spinal cord was externally normal. Histologically there was moderate loss of anterior horn cells at lower cervical and lumbar levels. There was a mild fibrillary gliosis throughout the anterior horns. The crossed pyramidal tracts showed loss of myelin that was not accompanied by gliosis. The anterior pyramidal tracts were well myclinated. In the posterior columns there was loss of myelin in the central parts of the tracts of Goll and in the ventro-medial parts of the tracts of Burdach. Fibrillary gliosis was conspicuous in the ventral half of the posterior columns. The anterior roots in the cervical and lumbar regions appeared more slender than usual but the axons in all roots were abundant and well myelinated.

The posterior root ganglia demonstrated degenerative changes in nerve cells with proliferation of capsule cells and formation of peripheral basket fibers in the cervical and lumbar ganglia. Swollen and beaded axons were also conspicuous in the affected ganglia and nodules of Nageotte were present. There was an inflammatory infiltrate consisting of lymphocytes and macrophages (Figure 3).

Examination of the peripheral nerves was unremarkable and axons of all sizes were abundant and well myelinated. Skeletal muscle showed a mild degree of non-specific wasting.

These findings are inconsistent with ALS, the archetype motor neuron disease, which does not include degeneration of the posterior columns as seen in this case. The find ings are best characterized as an atypical motor neuron syndrome occurring as a remote effect of oat cell carcinoma.

Anatomical diagnosis: Atypical motor neuron syndrome associated with oat cell carcinoma of lung.

Dr. Rex: As this man's hospital course progressed, considerable sus picion developed that a carcinoma was the underlying problem. He died at the VA hospital in early 1982, before chest CT was available there. I believe chest CT would now be the test of choice to image his lungs and mediastinum and would have demonstrated the mass. As it was, the mass was obscured on chest x-rays by progressively worsening left lung pneumonias. Five bronchoscopies with cytologic specimens failed to reveal evidence of the neoplasm. Oat cell carcinoma is a bronchogenic tumor but may spread almost completely

submucosally and thus may not produce an endobronchial mass. This is likely why the bronchoscopies and cytologies were negative in this case. Exploratory thoracotomy was contemplated late in his course but he was considered too ill to undergo it.

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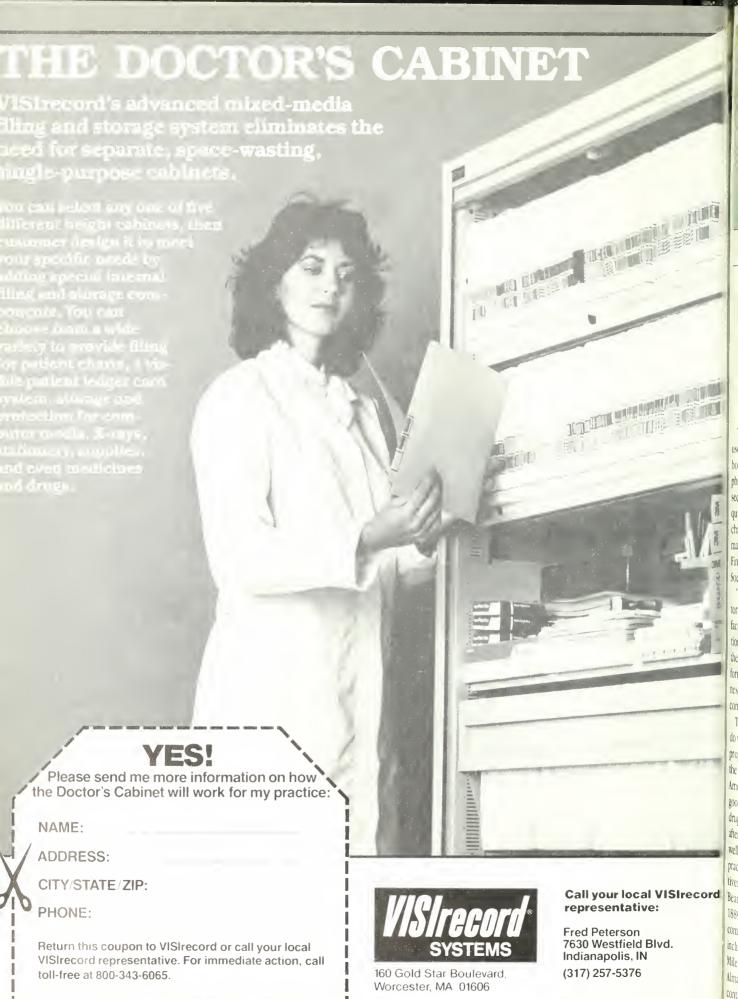
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Snakeroot Extract

Number 5

June, 1985

A NEWSLETTER OF INDIANA MEDICAL HISTORY

Miles Laboratories' Exhibit Opens at Society

The original television model of Speedy Alka-Seltzer® used eighty-five heads, hundreds of legs, and many other body parts to animate the character through stop-motion photography, involving up to 1,440 changes in a sixty-second commercial. Appearing first in 1952, Speedy quickly became one of America's best-known trademark characters. For fifty-four years, Alka-Seltzer® has remained the leading product of Miles Laboratories, Inc. Finally, Speedy will be visiting the Indiana Historical Society.

"Miles: The First Century" will open at the Indiana Historical Society exhibit hall on June 10, 1985. Using artifacts and business documents on loan from the collections of Miles' corporate archives, this exhibit illustrates the many successes and marketing ingenuity that transformed Franklin L. Miles' small proprietary medicine business into today's large, research-based pharmaceutical company.

The success of Alka-Seltzer® probably had as much to do with the advertising and marketing talents of the early proprietors of Dr. Miles' Medical Company as it did with the ubiquity of heartburn and indigestion among Americans. "Miles' Restorative Nervine" received such a good reception from Franklin Miles' patients and druggists that he was able to form his company in 1884, after fewer than ten years in practice. Miles himself was a well-trained eye and ear specialist, so his Elkhart, Indiana, practice could have flourished without tonics and calmatives. Businessmen George Compton and Albert Beardsley, however, joined the fledgling company in 1889, and quickly began to translate medical ideas into commercial success. Among the advertising artifacts included in the exhibit are Dr. Miles' Little Books, Dr. Miles' Weather Calendars, and Dr. Miles' New Weather Almanacs and Handbooks of Valuable Information, all containing tidbits of information to delight isolated farm families as the company advertised its medical products.



Speedy Alka-Seltzer epitomizes Miles' marketing flair. Photo courtesy Miles Corporate Archives.

From 1902 to 1942, some 40,000,000 of these publications were distributed throughout the country annually.

Along with Nervine, "Dr. Miles' Restorative Blood Purifier" (later the "Alterative Compound") and "Dr. Miles' Heart Cure" (containing digitalis) reached out to the neurasthenic, Progressive Era. By the 1920s, the company filmed commercials to follow on the coattails of

(continued on Page 3)

Museum Studies Restoration of Old Pathology Building

Restoring a historic structure is a complex, time-consuming, and expensive task. Many historical buildings have been altered drastically since their construction. Interiors often have been remodeled beyond recognition to accommodate a building's growing needs. Often there is little or no documentation on a building's original interior, making an accurate restoration impossible. Also, many historical buildings are structurally unsound, further complicating or halting restoration efforts. During the past year, the board of the Indiana Medical History Museum has studied the restoration of Central State Hospital's historic Old Pathology Building. Fortunately, the museum is not confronted with the problems described, but nonetheless faces several difficult restoration decisions.

The Old Pathology Building (originally called the Pathological Department of the Central Indiana Hospital for the Insane) was opened in 1896 as a research and teaching facility. The building was designed by Indianapolis architect Adolph Scherrer and was constructed by the John A. Schumaker Company. Ira Van Gieson, the director of the well-known Pathological Institute of the New York State Hospital, consulted on the design of the laboratories. The building had nineteen rooms, including four laboratories, an autopsy and dissection room, an anatomical museum, a library, and a teaching amphitheater. It was the hope of George F. Edenharter, M.D., then superintendent of Central State, that the building would provide the laboratories and necessary equipment to study the causes of mental illness.

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Snakeroot Extract is a joint publication of the Indiana Historical Society's Medical History Committee (315 West Ohio Street, Indianapolis, Indiana (6202) and the Indiana Medical History Museum (Old Pathology Building, 3000 West Washington Street, Indianapolis, Indiana (6222) The newsletter is mailed to members of both the committee and the museum

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Snakeroot Extract derives its name from the white snakeroot plant, a plant that is significant in Indiana medical history, for years, a mysterious disease called milk sickness plagued early Hoosiers. There were many theories as to the disease's cause, but the actual cause remained inknown until the 1920s. At that time, the disease was traced to the white snakeroot plant or, rather, to the consumption of milk from cows that had eaten it. The plant contains the poison tremetol.



The library in the Old Pathology Building at the turn of the century.

Moreover, Edenharter encouraged the area medical schools to use the building for classes in neurology and psychiatry. The building's research and teaching functions, as originally set forth by Edenharter, continued in effect until the 1960s. Medical school classes met in the building until 1956, and research on mental and nervous disorders continued until 1965.

Unlike most historic structures, the Old Pathology Building has escaped major architectural changes. The architectural firm of Richardson, Munson, and Weir, which recently performed a survey of the building, has termed the structure "Indiana's Rip Van Winkle building." The building is indeed a unique historical structure. Even though the building was in use until the 1960s, the laboratories were never modernized. The white oak woodwork, laboratory tables, and cabinets have remained as they were at the turn of the century. The solid oak amphitheater, too, has survived unscathed. The building has seen a few "reversible" changes. All the walls have been painted, and the stenciling which once adorned the tops of the walls in the building's library and reception room has been covered. Lighting fixtures were changed in the 1930s, as was the original floor covering. Linoleum protected the floors in the laboratories and work areas, whereas woven carpeting covered the floors in the library and reception room. The library and reception room were illuminated by chandeliers. Fortunately, there are photographs documenting the original condition of a number of these rooms.

Because relatively few changes have been made in the building, an "accurate" turn-of-the-century restoration would be possible. However, the question which arises is whether this type of restoration would be appropriate

Museum Studies Restoration

(continued from Page 2)

since much important research occurred in the building during the 1920s and 1930s. Undoubtedly, the restoration of the building to a particular time period will have to be approached with caution, lest a significant portion of the building's history be lost.

Another restoration concern of the museum is that the Old Pathology Building is not only a historical structure, but also houses a large collection of medical artifacts dating from the early 1800s. These artifacts require proper storage facilities, humidity and climate control, and exhibit space. Moreover, to care properly for the artifacts, as well as conduct the day-to-day business of the museum, office space and work areas are needed. But can all these needs be incorporated into a historic structure without sacrificing the historical integrity of the building? The firm of Richardson, Munson, and Weir carefully studied some of these problems and presented the museum board with some of their suggestions. Some of their solutions to the above problems include a heating system, which would utilize the building's existing steam radiators, and the remodeling of the historic structure's 2,200 square foot attic into a modern exhibition area. Yet, these solutions to the building's current needs also involve sacrifices of certain existing work areas to accommodate the heating and cooling system and stairwells to reach the third floor attic. Obviously, the board of the Indiana Medical History Museum will face these difficult decisions as it decides the best approach to the restoration of the Old Pathology Building.



The Old Pathology Building's reception room, ca. late nineteenth century.



The Old Pathology Building's histology laboratory, ca. late nineteenth century.

Miles' Exhibit Opens

(continued from Page 1)

feature movies. These commercials warned of "The Curse of Catharsis" and the help "Miles Anti-Pain Pills" could bring. The 1931 invention of Alka-Scltzer®, however, catapulted the company, and its second generation of directors, into the category of a modern pharmaceutical laboratory.

Miles Laboratories, Inc., created in the mid-1930s, introduced One-A-Day® vitamin products to drugstores of the 1940s. Bactine® antiseptic and mouthwash followed in the 1950s, at the same time Miles' expanding staff of chemists began to develop less commercially-oriented medical items. Clinistix® and Glucola® are probably two of the most familiar of these. Today's company employs 12,000 persons worldwide, with annual sales topping one billion.

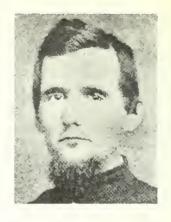
The exhibit was originally mounted in South Bend last May in observance of Miles' centennial. It was designed by Geoffrey J. Huys and Marsha Mullin of Discovery Hall Museum in South Bend. Miles' archivist Donald Yates, Ph.D., added some special objects and interpretive labeling for the move to Indianapolis. Miles maintains an active history program and archives. The archives, from which the exhibit materials were drawn, houses one of American industry's oldest collections of business records, advertising materials, and product samples.

Library Acquisitions

Samuel Orr of Evansville recently donated several rare medical volumes to the Indiana Historical Society Library. The books, from the library of Orr's great grandfather, Dr. Isaac Casselberry (1821-1873), are welcome additions to the Society's collections. Two of the works in the collection deserve particular mention: a first edition of John Redman Coxe's Practical Observations on Vaccination: Or Inoculation for the Smallpox (Philadelphia, 1802) and a third American edition of Alexander Hamilton's Outlines of the Theory and Practice of Midwifery (1797). Coxe (1773-1864) was the first physician to practice vaccination in the Philadelphia area and worked to overcome the strong popular resistance to this preventive measure by vaccinating his own infant son with vaccine matter from Edward Jenner. Coxe also taught pharmacy and chemistry at the University of Pennsylvania. Alexander Hamilton (1739-1802) was a professor of midwifery at the University of Edinburgh in the eighteenth century and helped establish the Lying-In Hospital at the university in 1791.

Dr. Casselberry was born in Posey County, Indiana, on November 26, 1821. He was a graduate of the University of Cincinnati Medical College and practiced medicine in Evansville. Casselberry took an active part in the Civil War, serving as a regimental surgeon for the First Indiana Cavalry and as Medical Director for the Fourth Indiana Division, the Third Indiana Cavalry Division, and the Medical District of Eastern Kansas. After the war, Casselberry re-

Indiana Historical Society Indiana Medical History Committee 315 West Ohio Street Indianapolis, IN 46202 Dr. 1saac Cassetberry (1821-1873)



turned to Evansville to practice medicine and served on the Evansville Board of Health and the School Board. In 1873, Casselberry was elected president of the Indiana State Medical Society, but died before serving his term.

Casselberry's interests extended beyond medicine to natural history and the history of medicine. Two of the books donated by Orr reflect this interest. A copy of the November, 1857, Nashville Journal of Medicine and Surgery contains an article by Casselberry entitled "Historical Review of Certain Causes of Epidemic Diseases." Bound with the 1852 proceedings of the third annual meeting of the Indiana State Medical Society is a publication of Casselberry's, A Description of Certain Fossil Bones Found Near Evansville, Indiana With Speculations Concerning the Animal to Which They Are Remains. Casselberry claimed that he had discovered the bones of a deinotherium magnum, or a Miocene or Pliocene mammal resembling a large elephant with tusks.



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ETHICS AND MEDICINE

2. The Hospital Medical Ethics Committee

STEPHEN E. OLVEY, M.D. Indianapolis

HE ADVANCES OF modern medicine have brought physicians many new and difficult decisions. Among the most challenging are when to discontinue patient eare and whether or not to institute CPR for selected patients. A patient who is mentally incompetent and terminally ill is one matter; one who is competent and no longer wishes to continue his or her struggle to survive is another.

There comes a point in everyone's life where cognitive function no longer exists or because of a disease process life no longer can be considered an asset, but rather a liability. At that point the physician, the patient if mentally competent, and the family often become embroiled in discussions involving that patient's terminal care. Too often, this decision-making process becomes confused, embittered, irrational, and inordinately prolonged.

Frequently, the family members themselves may be unprepared to par ticipate in conferences designed to reach important conclusions. They also may be reluctant to make decisions because of conflicting moral or philosophical biases within or among individual members. The physician, too, may possess personal, moral, theologic, or philosophical beliefs that make it

difficult for him/her to terminate or withhold therapy or to respond to patient or family requests to do so.

Presenting such special patient care problems to a group of peers and others especially knowledgeable and/or interested in such issues may assist in clarifying particular points for discussions, help put medical, personal, or social issues into proper perspective, and assist through group support and interaction—the—decision-making process.

Ethics Committees

Hospitals more and more are forming ethics committees or enlarging the role of their existing committees. Ethics committees have been in existence for many years but most often have been passive, discussing matters relating to internal medical affairs only. Ethical issues dealt more with the problems of fiscal responsibility and divisions of labor rather than the broader social issues. Questions on matters such as organ transplantation and genetic recombination only recently have been discussed. Not until the public brought to bear such issues as the right to die, the living will and wrongful death did hospitals and physicians realize that they could no longer simply discuss these issues among themselves, but that they must react to these issues by providing leadership, guidance, and compassion through an educational, albeit not binding, forum.

Structure

The structure of an ethics committee should be multi-disciplinary in nature and include not only hospital

employees but outside parties as well. Those physicians most often involved in ethical and moral judgments, i.e., intensivists, neurosurgeons, psychiatrists, transplant surgeons and neonatalogists, should be included. Included from the hospital also should be representation from social service, nursing, chaplaincy, as well as a member from the legal department and a representative from hospital administration. From outside the hospital, one should include an ethicist if available and/or a concerned lav person with experience and knowledge in ethical affairs. Local universities and/or seminaries are often a good source of outside, knowledgeable people. At Methodist Hospital in Indianapolis such a committee is currently operating and beginning to consider matters of active involvement.

Function

When an ethics committee takes an active role it must be free of any conflict of interest. For this reason people involved in bed utilization, DRGs, or who have strong biases regarding social and/or religious matters should not be involved. The topics of concern should be discussed and dealt with by virtue of their ethical considerations only. The committee, most importantly, cannot be adversarial or punitive in its relationship with the medical staff. An ethics committee, to be truly useful, must function as an advisory board available for presentation at any time. A large committee therefore can be subdivided into smaller representative groups so that consultation can be obtained on an immediate basis. In this

The author is Associate Director, Adult Intensive Care, and a member of the Hospital Ethics Committee, Methodist Hospital of Indiana, 1604 N. Capitol Ave., Indianapolis, Ind. 46206.

way, physicians, nurses, other hospital personnel, or family can bring ethical issues prospectively or retrospectively to the committee for discussion. No hard judgments should be rendered by the committee. Rather, the committee should function as an open forum to air ethical issues and form a majority opinion as to the best resolution of such issues.

Benefits

It is too soon to tell whether or not ethics committees are of benefit in handling these problems. The concept of an active ethics committee is too new for valid data to have been gathered. It stands to reason, however, that such a committee will increase the chances of arriving at an opinion which is not only rational but ethically valid in light of current thought. Benefits of such an ethics committee would include the following:

- The committee would determine that all of the relevant information has been obtained and communicated to the decision makers, and suggest additional sources of information where appropriate;
- The committee would identify the ethical issues as opposed to the emotional, legal, religious, or professional ones, and spell out the conflicting values, interests, and duties at stake;
- The committee would facilitate communication and help to resolve disagreements that are based on lack of information or on misunderstanding of facts or principles;
- The committee would provide support to staff and families by confirming the ethical complexity and, in some cases, the ethical acceptability of their decisions;
- The committee would recommend where appropriate that the hospital administration seek recourse to courts; for example, to have a guardian appointed for an incompetent patient, or to obtain a court order permitting certain treatment. Although the committee sometimes might precipitate judicial involvement, it would probably

avoid unnecessary recourse to litigation by aiding in the resolution of disagreements and by providing reassurance that decisions have been reached only after due consideration of the ethical factors, particularly the patient's best interest.

Pitfalls

Possible dangers do exist in the utilization of such a committee, however. The committee may, if not careful, bring inappropriate peer pressure to bear on individuals to make particular decisions, infringe on or subvert a family's or physician's prerogatives, violate a patient's right to confidentiality, diffuse the decisionmaking process so widely that no one feels responsibility for a final judgment, or the committee may become biased toward a given position. In addition, the ethics group may merely expand medical staff bureaucracy without functioning as originally intended.

Summary

Ethics committees with an active branch that stands ready to support, educate, and comfort the physician as well as the family in matters of patient care offers help in sorting out ethical considerations. By combining the physician and the family with a multidisciplinary committee composed of not only colleagues but clergy, ethicists, legal experts and social services, much of the indecision, guilt and frustration which arises in dealing with ethical affairs can be relieved. When these processes become entangled in ethical uncertainty the physician, the family, or other involved persons may consult the hospital's ethics committee for assistance. In this way, the patient's best interests can be served and the standards of ethical practice can be maintained.

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Hey, Maximus, Off Your Gluteus!

Commentary

HE RAPIDLY evolving concepts in alternative healthcare delivery systems in the United States are beginning to manifest a far-reaching effect on the psyche of the physician.

He used to be the high priest, almost almighty. No one dared question him or his judgment. The Ten Commandments, maybe, but never his judgment. He had at least between one to 10 years of postgraduate training, you know. And that made him perfect. Almost. So he was up there, on the pedestal.

He used to think that, as long as he was properly trained, and was practicing good medicine, he was safe up on that pedestal, untouchable.

But the ball game has changed. And still unfolding. The rules have been revised. The consumer is now the almighty. Not the physician. And it is no longer the product alone, or the integrity of the source of the product, that is most important. It is now the consumer, and what he wants, that counts most. And he wants the best medical care at the least cost possible, and right away!

So, down crumbles the pedestal. And the doctor and his image.

It is no longer business as usual. The past is indeed the past. It is history. It is NOW the future, yes, now, not tomorrow. And the sooner he stops his denial, the sooner he accepts this reality, the better it will be for his psyche and for the medical profession. And for his individual practice as well.

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PHILIP S. CHUA, M.D. Munster

Yes, it is no longer business as usual in many states. And the epidemic is spreading like wildfire all over the rest of the country. Even the blind could see that.

He, the almighty, the invulnerable, the arrogant, the chosen, the secure, shall be renamed plain and simple "healthcare provider." He shall now be more subject to be nailed to the Blue Cross, labeled imprudent by Prudential, charged inequitable by Equitable, and considered "minor" by Major Medical. He shall henceforth be confronted and confused by DRG, PRO, HMO, PPO, IPA, PPN, etc., even while eating his breakfast alphabet cereals. With cost-containment haunting him, quality assurance on top of him, with the new rules painted all over him, he is terribly confused, panicky and feeling very frustrated. Demoralized. Angry. And he is in a dilemma as to whether he should be HMOgenized or PPOed.

But the beast is still over-confident and too independent for his own good. He has a lot of self-denial. His glasses are tinted to shield him from the ugly color of the truth, the reality.

When a call for unity and solidarity is sounded by his colleague, who sees the storm and catastrophe coming, he thinks he is crazy and a false prophet. He refuses to see the writings on the wall—direct from California—that portends the fast approaching hurricane. he suspects the motive of his colleague who insists, "We, the physicians, must unite, organize ourselves and establish our own alternative plan now—otherwise enterpreneurs, or even Uncle Sam, might do it for us, which would be as disastrous as bilateral orchiectomies."

The physician is too individualistic, too independent, suspicious and paranoid for his own good and survival. He is also too busy to look and verify for himself the oncoming storm from the West. When a movement is organized, he then develops excessive phobia that the initiator might be in it only for the money. He disregards the obvious personal and financial sacrifices this particular colleague has made to accomplish an almost colossal task of organizing the medical community for its own survival. All he is concerned about are the possible negative aspects, disadvantages and risks of the program, without objectively equating these with the obvious positive virtues of the inevitable movement. He might even prefer to listen to misinformation

Dr. Chua is president and chairman of Comprehensive Healthcare Utilization Alternatives, Inc., a Preferred Provider Organization for northwest Indiana.

circulated by dissenters and detractors, and close his eyes to the facts. He might even go to the extent of indulging in petty and childish behavior of smearing posters and tearing down signs and announcements about the movement. Indeed, he suspects even the noblest of motives. At times, he even wishes the endeavor to fail. Yes, he would rather risk the future than trust his colleagues.

And this is why organized medicine is not fully effective and strong like labor unions in this country. And it is a pity. Organized medicine, had all the physicians truly supported it, could have been so powerful as to have prevented most of these dilemmas now haunting the physician.

Is it too late? I do not think so. I believe we could still salvage a great deal, if we act now. Therefore, let us not default by sheer indifference, neglect or suspicion, and unwittingly lose the control of our own destiny and our prerogatives to the other sectors of our community or to the government.

Let us be smart enough to trust our colleagues, and one another, establish better rapport with the consumers,

unite, organize, and act wisely and timely. Then, we could change the vision of the hole in a donut into a ring of bread around it. And deserve the bread.

Let us, therefore, not curse the darkness. Instead, let us light a candle to guide our own destiny for a brighter future. Let us transform this adversity into a great opportunity.*

Dr. Maximus, please, off your gluteus!

*Chua PS: PPO: Opportunity over Adversity. Indiana Medicine, 77(10):796-798, 1984.



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EDITORIALS

A Serious Look at Marketing Strategy

The Medical Practice Letter (The Monthly Newsletter on Medical Practice Management), published in New Haven, Connecticut, is now in its fifth year. It started as a well written and authoritative resource on the myriad of legal and social changes in the practice of medicine. Each issue has analyzed thoroughly each one of the many changes and new conditions, and has offered and discussed the options which physicians may adopt to improve the quality of medical practice and to avoid conditions likely to interfere with good quality medical practice.

A review of the issue of December 1984 is interesting. This issue discusses marketing in medical practice. Marketing has received somewhat of a cool reception in medical circles. The word sounds as though it is related to advertising and salesmanship. It is neither. No element of the process and art of marketing is contrary to medical ethics or professional conduct.

In essence, marketing is defined as the method of determining what consumers need and want, and then adapting or modifying to be able to provide their wants.

To quote *The Medical Practice Letter*: "Marketing is the process by which the needs of a specific group of potential consumers are identified and met. The basic marketing strategy is to look at products and services from the con-



sumer's point of view. Marketing strategy in health care is developed by analyzing patients' needs and designing services to meet them."

Marketing is especially important in today's practice of medicine. The approaching oversupply of physicians, the tendency of hospitals to project patient care outside the hospital, the development of free-standing emergency and out-patient clinics, the fad of "Doc in a Box," and similar developments to come, all present situations which are amenable to study and adaptation of principles involved for the private practice.

The success of all the above medical activities depends on whether or not they fill the consumers' needs and convenience.

(Individual copies of *The Medical Practice Letter* may be procured by writing to TMPL at 227 Everit St., New Haven, Conn. 06530. Back issues are priced at \$12. The annual subscription rate is \$135.)

Thank God, It's Friday

Letter to the Editor

Some time ago I sent you a letter concerning our Mother Tongue, which you were so kind as to publish. Since then I have had some ruminations upon retirement in which, as a fellow octogenarian, you might be interested.

If you always think of retirement as involving a fundamental change, you are right, and if you are dubious as to whether "this is for me," you are right again. The said change involves a gain in leisure time but a loss of duties. Duty is bound up with your identity, so if there is a loss of duty there is a corresponding loss of identity.

The impact of this will depend on how devoted you have been to your work (duty) compared to your other interests. For a while (perhaps, even indefinitely) your sense of passing time will be deranged because you have no duty-prescribed schedule. New "duties" will help but they likely will lack the urgency of the old ones.

There may well be a parallel here between aging and adolescence—with

a difference. Adolescence has a strong background of major hormonal changes, with increasing strength of both mental and physical powers, whereas in aging these powers dwindle. For even the mental powers not to dwindle is the exception, not the rule.

For the retiree to cope with this situation is often difficult, but he can help himself by quotas of accomplishment (pseudo-duties?) in areas he can handle.

Even then, how can he make any progress in a field full of younger, more forceful people? The answer has appeared in the formation of groups, such as 50-year clubs, the A.A.R.P., etc. Since there is a sort of secondary threshold at age 80, I am about to propose the formation of another A.C.O.G.-the American College of Octo-Genarians. This should not upset the American College of Obstetricians and Gynecologists because the obstetricians are already beginning to retreat before the onslaught of litigation. We Octos can join the chorus of "Thank God, It's Friday." - Alister Caobhain, Route 13, Box 31.

The Pharmacist's View of Physician Drug Dispensing

Guest Editorial

The patient "pool" has remained relatively stable although the number of physicians has shown a sharp increase during the past 10 years. This increase in the supply of physician manpower has largely benefitted the public. The patient's access to a doctor's office has become easier, as evidenced by the move of more physicians to residential neighborhoods and shopping centers. But for pharmacists, it might be the beginning of a new competitive outlet.

In the past, most physicians did not dispense drugs to their patients because it could not be economically justified. The gross margin from dispensing was certainly lower than the professional fee from patient consultations and diagnoses. But now, the number of patient visits can shrink to

a point where drug dispensing may become an attractive alternative to recovering costs of operating a physician's office.

In a free enterprise system, if a physician can dispense drugs more efficiently and the patient can benefit from physician dispensing, we do not see many legal problems. We are skeptical about the physician doing the pharmacist's job. Furthermore, the freedom of choice is denied to the patient. We wonder how many patients would insist on getting a product from the physician when the physician orders to the contrary.

But there is a job we have to do to protect our turf. Ask yourselves: Why should a patient get his prescription filled in my pharmacy when there is no perceived difference in services offered by the physician's office and my pharmacy? If there is a difference, how come the patients do not know and appreciate it? Have I told them what I do for them in my role as a health professional?

Our knee-jerk response to the problem of physician dispensing could have been, "Let's band together and ask the state's legislative body to pass a law to prohibit physicians from dispensing drugs to their patients." But this is just a palliative measure. The real job is to "show and tell" the patients what you do for them, how it benefits their health, and why your services are valuable.

In the final analysis, it is the consumer who will dictate the market-place. If we meet the consumer's need for health-related products and services, remain flexible to change with the time, our place in the marketplace is assured. It is also the time for asking some questions, e.g., Is your drug wholesaler selling drugs to dispensing physicians in your trade area? If so, why? Further, are you patronizing the same mail order generic firms that are

also selling (often at lower prices) their drug products to the physicians?

Problems have to be solved. They just don't go away with the passage of time. Prevention or early detection and treatment helps more than anything else. If your prescription-filling activity shows an unexplainable decline, check to see if you have a dispensing doctor in your area. One suggested way is to call the physician, explain your side of the story, even channel some patients to him to prove your point; but never forget the most important person—the patient.—"Action in Pharmacy" newsletter, April 1985

ISMA Constitutional Amendment

As required by Article X (Amendments) of the ISMA Constitution, INDIANA MEDICINE announces the following Constitutional amendment:

Resolution 84-6 (Subject: Article III—ISMA Constitution—Component Societies) was introduced by the ISMA Commission on Constitution and Bylaws during the 1984 session of the House of Delegates. The resolution, which follows, was adopted. The boldface type indicates changes to the present language.

"Resolved, That Article III—Component Societies, be amended to read: Component societies are those county, district or other medical societies as specified in the bylaws contained within the state of Indiana, and who hold charters from this Association."



AUXILIARY REPORT

Muriel Osborne (Mrs. John) ISMA Auxiliary President 1985-86

This year's County Reports again heralded the fact that medical spouses are truly active, involved, energetic individuals who are committed to supporting the areas of concern the ISMA and the ISMA-Auxiliary believe vital if we are to promote the image of the physician as a friend. In all counties there was evidence of community enrichment due to auxiliary involvement. For example:

ALLEN COUNTY: Gives five \$400 nursing scholarships; supports *Daybreak*, a child abuse center, with a "Diapers for Daybreak" project; raised \$3,500 to purchase equipment and medications for the Three Rivers Neighborhood Health Services Clinic.

BARTHOLOMEW-BROWN COUNTY: Screened 450 pre-schoolers for "Lazy Eye."

CLARK COUNTY: Joined forces with Floyd County to sponsor public service announcements on television and radio "To Promote the Positive Aspects of Aging."

DELAWARE-BLACKFORD

COUNTY: Divided the \$3,200 profit from their sharing card equally between AMA/ERF and the local Hospitality House, which is one of seven such houses in the U.S.

ELKHART COUNTY: Sponsored two scholarships in the amount of \$1,500 each so that county girls could continue their education in medically related fields.

FLOYD COUNTY: Has ongoing Pediatric Playroom Project and contributed \$950 to their local Memorial Hospital for the establishment of an OB Newsletter program; contributed to Hospice and to Hedden House, a community halfway house for recovering alcoholic women.

GRANT COUNTY: Gave six scholarships to county students pursuing a medically related career; began an

524

ongoing project to donate linens and toiletries to be used as bedside care packets in hospitals in Haiti.

KNOX COUNTY: Auxilians aided AMA/ERF, the Pediatric Dept. of Good Samaritan Hospital and continued to teach and co-sponsor the K.I.S.S. Project with the American Red Cross.

LAKE COUNTY-HAMMOND: Collected surplus or outdated medical supplies for World Medical Relief; donations from a holiday sharing party totaled \$1,600 with which eight families were helped, as well as the local home for battered women and the hospital social service emergency fund.

MARION COUNTY: Awarded \$6,400 in scholarships to seven nursing students and donated \$1,000 to the I.U. Medical Student Loan Fund; continued to work with the Love Seat Project at Methodist Hospital, the Ronald McDonald House and with the medicine collection for Haiti.

NOBLE-LA GRANGE COUNTY: This 12-member organization contributed \$811.46 to AMA/ERF through sales of jewelry, wooden hobby horses, stationery and Christmas cards and a county Sharing Card.

ST. JOSEPH COUNTY: Concentrated on members' education to stimulate interest; provides help to the Blood Bank twice weekly during the winter months in recording and screening donors.

TIPPECANOE COUNTY: Donated \$320 to Group Homes, Inc., which operates four juvenile care homes in Lafayette; a scholarship auction netted \$1,009.50 to help needy students seeking degrees in nursing; supplies and operates a Gift Cart at Tippecanoe Villa on a bimonthly basis.

VANDERBURGH-SOUTHWEST-ERN COUNTY: Members made the puppets and stage for a puppet show about child abuse, which was presented at 10 elementary schools; combined proceeds from the holiday sharing card, cook book and spring flower sales and an auction raised \$12,000, to be contributed to community health care organizations.

VIGO COUNTY: Continues their involvement with Meals-on-Wheels and participates in the blood screening program conducted by the American Heart Association; provides three Indiana State U. Nursing students scholarships of \$300 each.

WABASH COUNTY: Underwrites the infant and toddler safety seat program at the Wabash County Hospital, where 240 seats are now in circulation; members are assisting the county medical society with the SADD program.

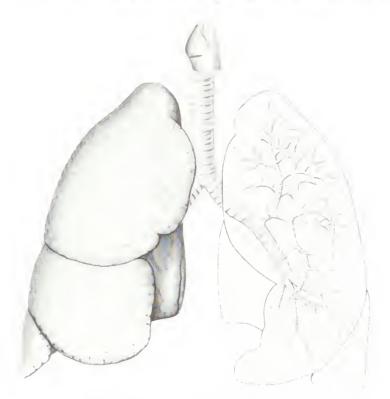
WAYNE-UNION COUNTY: Assesses each member \$10 for a scholarship fund in addition to their dues payment; netted \$1,500 in the sixth annual "Style Show, Cards and Games, Salad Luncheon" where proceeds are channeled to the I.U. East, Nursing School.

WELLS COUNTY: Members completed a Christmas quilt, which was the prize in a raffle that raised \$1,000 to be used as a scholarship for students in medically related fields.

M.A.L.s: 51 auxilians who are Members-At-Large are dues paying members of ISMA-Auxiliary; special emphasis has been made to recruit spouses of ISMA delegates to join M.A.L. ranks.

The 1985-86 ISMA-Auxiliary year has now begun! Muriel Osborne was installed as our president on April 25. Muriel challenges us "To Reach For The Stars"—the stars of auxiliary concern—AMA/ERF, HEALTH CAREERS, LEGISLATION and MEMBERSHIP!.—Martha Stout, Editor, "The Pulse"

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ment should include sigmoidoscopy appropriate bacteriologic studies, and fluid etechnolyte and protein supplementation When the collist does not improve after the drug has been up discontinued or when it is severe oial vancomprin is the drug of choice for antibiotic associated pseudomembanous collist produced by C difficile. Other causes of collist should be ruled out.

colitis

Usage in Pregnancy — Pregnancy Category B — Reproduction
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Note: Ceclor * cetaclor Etilyr is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to peniculin-allergic patients.

Peniculin is the usual drug of choice in the treatment and prevention of strepticocal infections including the prophylaxis of rheumatic News See prescribing information.



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NEWS NOTES

ISMA Producing TV Series on Aging

A series of television programs designed to provide information to senior citizens about how to maintain a healthy lifestyle is being produced by the ISMA.

The six-part series, "Healthy, Happy and Wise," went into production in April.

Twelve Indiana senior adults have been selected to "star" in the programs, which will include segments on planning for aging; fitness; fears and stress; drugs, devices and healthy practices; support relationships; and high-tech medicine.

The series will be aired over public broadcasting stations this fall.

Return of 'The Brain'

The Ciba-Geigy Corporation is making available to all 26,000 of the nation's high schools a special study guide for science classes to be used in conjunction with the nationwide rebroadcast this fall of the public television series, "The Brain."

The guide contains teachers' and students' materials. It is easily duplicated and may be easily incorporated into existing curricula.

"The Brain," which took five years to prepare, is presented as an eightpart series that examines the past, present and future of brain science.



"After taxes, you have to pick yourself up, dust yourself off, and start all over again!"

ISMA Supports HADD

The ISMA is contributing \$10,000 to Indiana's battle against drunk driving.

The contribution, approved this spring by the Board of Trustees, was in response to a request for ISMA to co-sponsor the Hoosiers Against Drunk Driving conference scheduled for Aug. 17-18. The request was introduced by Marion County Prosecutor Steve Goldsmith and Dr. Michael DuBois, representatives of the Governor's Task Force to Reduce Drunk Driving.

ISMA's financial contribution will sponsor 200 high school students who will meet to determine how they can help reduce drunk driving in the state. County medical societies are being asked to nominate and sponsor students to attend the conference.

More Field Coverage

As a result of recent action by the ISMA Board of Trustees, a third field staff representative has been appointed. He is Bob Sullivan, former ISMA director of communications, who joins Howard Grindstaff and Sara Klein in serving as the "eyes and ears" of the Association in its effort to enhance two-way communication at the grass roots level.

Sullivan is assigned to the central and east central areas of the state (Medical Districts 6, 7 and 8). Grindstaff retains responsibility for northern and north central areas (Medical Districts 9, 10, 11, 12 and 13), while Klein covers the southern and west central areas (Districts 1, 2, 3, 4 and 5).

PCF Surcharge May Rise

As a result of new legislation affecting the Indiana Medical Malpractice Act, the annual surcharge levied on the premiums of all health care providers in Indiana may be increased to 75%. The increase, which may be assessed immediately, is intended to ensure the fiscal solvency of the Patients' Compensation Fund.

After Jan. 1, 1986, the surcharge may be increased up to 100% at any time when the PCF balance is less than \$15 million.

More information on this and other changes in malpractice legislation was contained in an April 24 letter sent to ISMA members by Dr. Lawrence E. Allen, president.

News from the AMA

- A second printing of "Physician's Cost Containment Checklist" is now available. The initial press run of 7,000 copies was depleted within two months, and the AMA has received orders for 26,000 additional copies. The nine-page booklet describes practical ways physicians can reduce costs in everyday encounters with patients. Single copies are free. For more information, contact the Dept. of Health Care Financing and Organization, AMA Headquarters—(312) 645-4868.
- State licensing boards are now being alerted by the AMA when licensure action has been taken against a physician in other states. The new procedure identifies physicians who, having been disciplined in one state, may attempt to practice in another jurisdiction where they hold a license. The AMA is using its computerized Physician Masterfile to speed communications between licensing bodies.
- Eleven state medical associations increased their representation in the AMA House of Delegates, adding 13 members to the policy-making body. Pennsylvania and Texas increased their delegations by one each because of growth in the number of AMA members in those states. The other states increased their delegate counts through a 1984 bylaw amendment that allocates one additional delegate if 75% or more of a state association's members also belong to the AMA and two additional delegates if 100% of the association's members also belong to the AMA. Illinois and Oklahoma gained two delegates each, while Arizona, Iowa, Nebraska, North Dakota, Ohio, Tennessee and Wisconsin gained one delegate each.

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For the Asking . . .

- The American Hospital Association has a new edition of A Strategic Planning Process for Hospitals, by Joseph P. Peters. It is described as giving the newest and best thinking on strategic planning, much of it from corporate America. The book is published by American Hospital Publishing, Inc., which also offers Guide for Preparation of Constitution and Bulaws for General Hospitals; Hospital Ambulatory Care: Making It Work; Productivity and the Quality of Work Life in Hospitals; Ambulatory Health Care Evaluation; Principles and Practice; and Improving Management Performance in Health Care Institutions: A Total Systems Approach. For details, write to American Hospital Publishing, Inc., 211 E. Chicago Ave., Chicago 60611.
- "Jobs for Disabled People" is the title of the latest Public Affairs Committee Pamphlet (No. 631). It is written by Frank Bowe, who explains federal laws that assure the rights of the disabled to education, training and

jobs. He says there are sound reasons why disabled workers are desirable, explaining that they are more loyal, do better work and are off for sickness less when compared to average ablebodied workers. The pamphlet may be obtained for \$1 from the Public Affairs Committee, 381 Park Avenue South, New York, N.Y. 10016.

• "Diet and Cancer" is the title of a 36-page booklet published by the American Council on Science and Health, which reports on various scientists and researchers who have adopted the attitude best expressed by "wait a minute," "not so fast," and "let's take a more careful look before jumping to conclusions." The author of the report, Dr. Michael W. Pariza of the University of Wisconsin, says the current skirmish over dietary fiber "is an excellent example of the very tentative 'state of the art' in this field." For a complimentary copy, send a self-addressed, stamped (39°), business-size (#10) envelope to Diet and Cancer Report. ACSH, 47 Maple St., Summit, N.J. 07901.

RMS Election Results

Eight resident physicians have been elected to serve as Resident Medical Society officers for 1985-86, beginning July 1. They are:

Dr. John G. Terry, Indianapolis, I.U. Medical Center, president and AMA and ISMA alternate;

Dr. Stephen Coon, Indianapolis, I.U. Medical Center, president elect and AMA alternate;

Dr. Lana Patch, Beech Grove, St. Francis Hospital, secretary-treasurer and AMA delegate;

Dr. Paul Daluga, Jr., Indianapolis, St. Francis Hospital, AMA delegate; Dr. Silvio Garcia, Speedway, I.U. Medical Center, AMA delegate;

Dr. Mark Hochstetler, Indianapolis, St. Francis Hospital, AMA delegate; Dr. Daniel Walters, Muncie, Ball Memorial Hospital, AMA alternate;

Dr. Steven Lester, Speedway, I.U. Medical Center, ISMA delegate and AMA alternate.

Physician Recognition Awards -



The following ISMA physicians are recent recipients of the AMA's Physician Recognition Award. This award is official documentation of Continuing Medical Education hours earned, and is acceptable proof in most states requiring CME in re-registration that the mandatory hours of CME have been accomplished.



Andrew, Jerald L., Fort Wayne Asher, James W., Indianapolis Bierlein, Alan H., LaPorte Booth, Franklin M., South Bend Buchman, Marshall H., New Albany Bullington, George E., Franklin Carroll, Mary E.D., Crown Point Clements, Robert E., Greenfield Crates, Gordon C., Denver Curran, William L. Jr., Jasper Drummond, James A., Milan Farrell, John J., Greenfield Fisher, Thomas H., Indianapolis Foley, Phillip D., Middletown Fuller, Robert G., Columbus Gailani, Salman D., Munster Gehring, Gordon G., Vincennes Gentleman, James W., Crown Point Gray, Kenneth L., Speedway Hanke, Carl W. III, Indianapolis Hoover, Joseph R., Fort Wayne Horner, Terry G., Indianapolis Hughes, William B., Waterloo Koontz, James A., Vincennes Langston, Edward L., Flora Liebner, Michael S., Logansport Lovett, Harvey D., Zionsville Macri, Paul A.C., Mishawaka Manifold, Harold M., Bloomington Milan, Joseph F., Bloomington Moayad, Cyrus, Valparaiso Nale, Stephen W., New Albany Nicely, Paulette G., Indianapolis Nill, John H., Fort Wayne O'Brien, Francis E., Rensselaer Overley, Toner M., Indianapolis

Patel, Kantilal K., Connersville Pontaoe, Alejandro G., Evansville Porcaro, Joseph P., Anderson Rowand, Randall W., Rising Sun Sala, Walter R., Merrillville Scamahorn, Malcolm O., Pittsboro Shapiro, David A., Monticello Sharp, Thomas W., Bloomington Slack, John D., Indianapolis Stewart, John C., Kokomo Stine, Marshall E., Bremen Stratigos, Joseph S., South Bend Stucky, Mitchell B., Fort Wayne Swaim, J.F., Rockville Tadatada, Victoriano J., Salem Tower, James H., Shelbyville Wells, William R., Princeton Yoder, Steven M., Goshen

news notes

Here and There . . .

Dr. Panayotis G. Iatridis of Gary, Dr. Ali O. Kheirbek of East Chicago, Dr. Stephen D. McMurray of Fort Wayne, and Dr. Robert T. Woodburn of Merrillville are newly elected fellows of the American College of Physicians.

Dr. John C. Jarrett of Indianapolis recently served as a visiting professor at Bloomington Hospital, lecturing on "Infertility In Vitro Fertilization."



Dr. Khalouf

Dr. Herbert C. Khalouf of Marion will fill the unexpired term of the late Dr. Arvine Popplewell as an AMA alternate delegate.

Dr. Clarence E. Ehrlich of Indianapolis recently delivered two lectures at the University of Alabama on cytoplasmic receptors and surgery and chemotherapy for ovarian cancer.

Dr. Jerry L. House of Indianapolis discussed "Primary Central Nervous System Tumors in the Cerebellopontine Angle" at an international symposium on neurological surgery of the ear and skull base, held recently in Sarasota, Fla.

Dr. Virginia M. Wagner of Indianapolis received the Marion County Cancer Society's Little Red Door Recognition Award in April; she helped establish Camp Little Red Door, an an nual summer camp for children with cancer.

Dr. Richard A. Tibbals of Evansville discussed oral cancer at a spring

meeting sponsored by the Dubois County unit of the American Cancer Society.

Dr. Cherryl G. Friedman of Noblesville discussed "How to Keep Smiling When Your Husband Retires" during an April women's luncheon series sponsored by Riverview Hospital.

Dr. Frank Walerko and Dr. Douglas J. Wilson conducted classes at St. Joseph Hospital, South Bend, in April for the early detection of testicular cancer.

Dr. William D. Dannacher of Wabash discussed various forms of cosmetic surgery on a local television program in April.

Dr. Robert K. Ellis of Elkhart has been elected to membership in the American Society for Surgery of the Hand.

Dr. Philip J. Ryan of Indianapolis was guest speaker at the April meeting of the Madison County Chapter, American Diabetes Association.

Dr. Walter J. Filipek of South Bend discussed chronic obstructive lung disease during the April meeting of the South Bend COPE Club.

Dr. Tae G. Kiehm of Mishawaka was guest speaker at the April meeting of the local Hypoglycemia Support Group.

Dr. John S. Rodway of Columbus discussed preventive health maintenance at a spring meeting at Jackson County Hospital for Families Facing Cancer.

Dr. William F. Nowlin of Merrillville addressed the April meeting of the I Can Cope group at St. Mary Medical Center, Gary.

Dr. Mark L. Dyken of Indianapolis is the new chairman of the American Heart Association's Stroke Council.

Dr. John T. Munshower of Indianapolis participated in a panel discussion at the April meeting in Indianapolis of the Parkinson's Awareness Association.

Dr. Larry D. Lovall and Dr. Garnet R. Harris of Danville recently conducted classes on drug abuse for Sixth Grade pupils at Pittsboro Elementary School.

Dr. George T. Lukemeyer of Indianapolis has been elected to a second term as secretary general of the American College of Physicians.



Dr. Vinicor

Dr. Frank Vinicor of Indianapolis presented an update on diabetes research at the April meeting of the Henry County American Diabetes Association.

Dr. Thomas J. Cittadine, a Noblesville orthopedic surgeon, discussed athletic injuries at an April public education meeting sponsored by Riverview Hospital.

Dr. John C. Peterson of Muncie discussed pre-menstrual syndrome during an April PMS seminar sponsored by Ball State's Minnetrista Center.

Dr. Ronald R. Elder, Dr. Mark C. Jones and Dr. Thomas P. Krueger of Evansville discussed "Aching Backs" at an April community health forum sponsored by Deaconess Hospital.

Dr. James C. Harris of Indianapolis has been elected associate medical director of American United Life.

5 Students Head MSS

Five Indiana University School of Medicine students will serve as the governing council for the newly formed ISMA Medical Student Society. The new 1985-86 officers are:

Dean Beckman, student delegate, Indianapolis: Eva Fadul, alternate delegate, Indianapolis; Doug Zale, chairman, Indianapolis; Bob Flint, vice-chairman, Indianapolis; and Anita Spitz, secretary-treasurer, Speedway.

New ISMA Members

The following physicians were welcomed in March as new members of the Indiana State Medical Association:

William F. Armstrong, M.D., Indianapolis, cardiovascular diseases.

George R. Aronoff, M.D., Indianapolis, clinical pathology.

Bryan Ashton, D.O., Warsaw, otorhinolaryngology.

John C. Bailey, M.D., Indianapolis, cardiovascular diseases.

Shari L. Barrett, M.D., Evansville, dermatology.

Kenneth W. Beckley, M.D., Indianapolis, nephrology.

James A. Berndt, M.D., Bremen, family practice.

James D. Buck, M.D., Indianapolis, internal medicine.

Brent C. Burke, M.D., Indianapolis, anesthesiology.

Christina Campbell, M.D., Indianapolis, anesthesiology.

Michael W. Chitwood, M.D., Indianapolis, family practice.

Philip A. Christiansen, M.D., Indianapolis, gastroenterology.

Carol A. Clark, M.D., Anderson, obstetrics and gynecology.

Dewey J. Conces, M.D., Indianapolis, diagnostic radiology.

Betty C. Corya, M.D., Indianapolis, cardiovascular diseases.

David W. Crabb, M.D., Indianapolis, gastroenterology.

James E. Currier, M.D., Anderson, therapeutic radiology.

Paul Daluga Jr., M.D., Indianapolis, family practice.

John W. Deppe, M.D., Evansville, orthopedic surgery.

James C. Dillon, M.D., Indianapolis,

internal medicine.
Carol D. Farr, M.D., Indianapolis,

anesthesiology.

David L. Farr, M.D., Indianapolis, anesthesiology.

Harvey Feigenbaum, M.D., Indianapolis, cardiovascular diseases.

Ardhendu Ghosh, M.D., Terre Haute, obstetrics and gynecology.

Deborah C. Givan, M.D., Indianapolis, pulmonary diseases. John D. Graham III, M.D., Beech Grove, cardiovascular diseases.

Richard S. Graul, M.D., Evansville, pathology.

Robert F. Guthrie, D.O., Munster, emergency medicine.

James J. Heger Jr., M.D., Indianapolis, cardiovascular diseases.

Eric A. Henricks, M.D., Indianapolis, anesthesiology.

John C. Hilgenberg, M.D. Indiana polis, anesthesiology.

Richard A. Hirschler, M.D., Syraeuse, family practice.

Linda A. Huck, M.D., Indianapolis, internal medicine.

James E. Jarrett, M.D., Indianapolis, obstetrics and gynecology.

James D. Kasten, M.D., Indianapolis, obstetrics and gynecology.

Dennis E. King, D.O., Vincennes, radiology.

Martin B. Kleiman, M.D., Indianapolis, infectious diseases.

Suzanne B. Knoebel, M.D., Indianapolis, cardiovascular diseases.

David A. Kovach, M.D., Indianapolis, anesthesiology.

Gopal Krishna, M.D., Indianapolis, anesthesiology.

Kevin J. Lavelle, M.D., Indianapolis, internal medicine.

Byung I. Lee, M.D., Indianapolis, neurology.

Glen A. Lehman, M.D., Indianapolis, gastroenterology.

Lawrence Lumeng, M.D., Indianapolis, gastroenterology.

Douglas R. Maxwell, M.D., Indianapolis, nephrology.

Richard L. McCammon, M.D., Indianapolis, anesthesiology.

Philip F. Merk, M.D., Indianapolis, pediatrics.

William M. Miles, M.D., Indianapolis, cardiovascular diseases.

Stephen N. Morris, M.D., Indianapolis, cardiovascular diseases.

John M. Morse, M.D., Terre Haute, gastroenterology.

Lois L. Moss, M.D., Lawrenceburg, pediatrics.

Daniel A. Neumann, M.D., Indianapolis, general surgery.

Randall G. Norris, M.D., Jasper, orthopedic surgery.

Jacqueline A. O'Connell, M.D., Indianapolis, cardiovascular diseases.

Katherine W. O'Connor, M.D., Indianapolis, cardiovascular diseases.

Donald P. Orr, M.D., Indianapolis, adolescent medicine.

Michael G. Orr, M.D., Indianapolis, ophthalmology.

Andre-J Ouellette, M.D., Holland, family practice.

Lillie-Mae M. Padilla, M.D., Indianapolis, obstetrics and gynecology.

Murray H. Passo, M.D., Indianapolis, rheumatology.

Michael W. Peters, M.D., Evansville, emergency medicine.

David V. Poer, M.D., Indianapolis, ophthalmology.

Arthur J. Provisor, M.D., Indianapolis, pediatric hematology-oncology.

Eric N. Prystowsky, M.D., Indianapolis, cardiovascular diseases.

Chalapathi C. Rao, M.D., Indianapolis, anesthesiology.

Ken O. Ridgeway, M.D., Greentown, family practice.

Larry D. Rowe, M.D., Wolcottville, family practice.

Edwin J. Smith, M.D., Indianapolis, nephrology.

Ernest E. Smith, M.D., Indianapolis, pediatrics.

Frank A. Snyder, M.D., Bremen, family practice.

Gust T. Spenos, M.D., Indianapolis, neurology.

Fred Spottsville Jr., M.D., Anderson, cardiovascular diseases.

Leon Stein, M.D., Indianapolis, internal medicine.

Robert B. Stonehill, M.D., Indianapolis, internal medicine.

Jan Swanson, D.O., Indianapolis, internal medicine.

Robert S. Tepper, M.D., Indianapolis, unspecified.

Venkatachala N. Vitalpur, M.D., Fort Wayne, internal medicine.

Rebecca S. Wappner, M.D., Indianapolis, pediatrics.

Robert M. Weetman, M.D., Indianapolis, neoplastic diseases.

Eric S. Williams, M.D., Indianapolis, internal medicine.

Thomas M. Wolfe, M.D., Indianapolis, anesthesiology.

James C. Wright Jr., M.D., Indianapolis, pediatrics.

Kenneth A. Young, M.D., Beech Grove, unspecified.

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CME QUIZ-

TO OBTAIN ONE HOUR OF CATEGORY 1 AMA CME CREDIT, answer the following questions by circling the correct answer on the answer sheet below. Complete and clip the application form and mail it to: Indiana University School of Medicine, CME Division, Fesler Hall 224, 1120 South Dr., Indianapolis 46223.

Acquired Immunodeficiency Syndrome

CONTINUED FROM PAGES 459-465

- 1. More than 70% of the cases of AIDS are in which one of the following groups?
 - a. Intravenous drug abusers.
 - b. Transfusion recipients.
 - Sexually active homosexuals and bisexual men with multiple sex partners.
 - d. Health care workers.
- 2. Most hemophiliacs with AIDS have been treated with:
 - a. Cryoprecipitate.
 - b. Factor-VIII concentrate.
 - c. Blood.
 - d. Fresh frozen plasma.
- 3. Opportunistic infections occurring in AIDS patients are related to:
 - a. Hypergammaglobulinemia.
 - b. Cytomegalovirus infections.
 - c. Defects in cell-mediated immunity.
 - d. Homosexual lifestyles.
- 4. The etiology of AIDS is strongly linked to:

- a. Cytomegalovirus.
- b. Epstein-Barr virus.
- c. HTLV III virus.
- d. Homosexual lifestyles.
- 5. Diagnosis of AIDS is presently made by:
 - a. HTLV III antibody testing.
 - b. Lymphadenopathy in an ill homosexual.
 - Finding an opportunistic infection or neoplasm in an otherwise seemingly normal host.
 - d. Abnormal T-lymphocyte studies.
- Which of the following is not true about Pneumocystis carinii pneumonia in AIDS patients;
 - a. Insidious onset of cough, dyspnea and fever occur.
 - b. Arterial oxygenation and chest x rays may be normal.
 - c. It is the most common opportunistic infection.
 - d. Side effects from trimethoprim sulfamethoxazole are uncommon.

- 7. AIDS is presently managed by:
 - a. Suramin.
 - b. Treating complicating infections and neoplasms.
 - c. Interferon.
 - d. Interleukin 2.
- Which of the following recommen dations to prevent the spread of AIDS is incorrect:
 - a. High risk group members should not donate blood.
 - Avoid sexual contact with persons known or suspected of having AIDS.
 - c. Blood transfusions should be given only when medically necessary.
 - d. High risk group members should not work in health care institutions.
- The risk of transmission of the virus causing AIDS to health care workers, including needle stick injuries is:
 - a. Small, if any.
 - b. Prevented by gamma globulin.
 - c. Moderate.
 - d. 100%
- The recommended isolation precaution for hospitalized AIDS patients is:
 - a. Strict isolation.
 - b. Respiratory isolation.
 - c. Blood/body fluid precaution.
 - d. Contact isolation.

MAY CME QUIZ Answers

Following are the answers to the CME quiz that appeared in the May 1985 issue: "Polycythemia in the Newborn Infant," by D. Wade Clapp, M.D., et al.

- 1. d 2. c 3. d 4. c
- 7. a 8. d 9. c 10. b

- Answer sheet for Quiz: (AIDS . . .)
 - 1. a b c d
 2. a b c d
 3. a b c d
 4. a b c d
 5. a b c d
 7. a b c d
 9. a b c d
 10. a b c d

I wish to apply for one hour of category 1 AMA Continuing Medical Education credit through the I.U. School of Medicine. I have read the article and answered the quiz on the answer sheet above. I understand that my answer sheet will be graded confidentially, at no cost to me, and that notification of my successful completion of the quiz (80% of the questions answered correctly) will be directed to me for my application for the Physician's Recognition Award of the American Medical Association. I also understand that if I do not answer 80% of the questions correctly, I will not be advised of my score but the answers will be published in the next issue of Indiana Medicine.

Name (please print or type)

Address

Identification number (found above your name on mailing label)

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To be eligible for this month's quiz, send your completed, signed application before July 10, 1985 to the address appearing at the top of this page.

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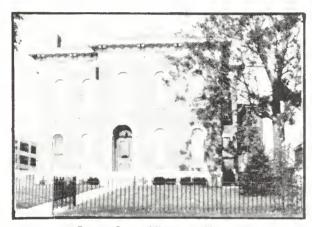
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References: 1. Kales J et al: Clin Pharmacol Ther 12:691-697, Jul-Aug 1971. 2. Kales A et al: Clin Pharmacol Ther 18:356-363, Sep 1975. 3. Kales A et al: Clin Pharmacol Ther 19:576-583. May 1976. 4. Kales A et al: Clin Pharmacol Ther 19:576-583. May 1976. 4. Kales A et al: Clin Pharmacol Ther 32:781-788, Dec 1982. 5. Frost JD Jr., DeLucchi MR. J Am Geriatr Soc 27:541-546, Dec 1979. 6. Kales A, Kales JD Jr. Clin Pharmacol 3:140-150, Apr 1983. 7. Greenblatt DJ., Allen MD., Shader RI: Clin Pharmacol Ther 21:355-361, Mar 1977. 8. Zimmerman AM. Curr Ther Res 13:18-22, Jan 1971. 9. Amrein R et al. Drugs Exp. Clin Res 9(1):85-99, 1983. 10. Monti JM. Methods Frind Exp. Clin Pharmacol 3:303-326, May 1981. 11. Greenblatt DJ et al: Sleep 5(Suppl 1):S18-S27, 1982. 12. Kales A et al: Pharmacology 26:121-137, 1983.

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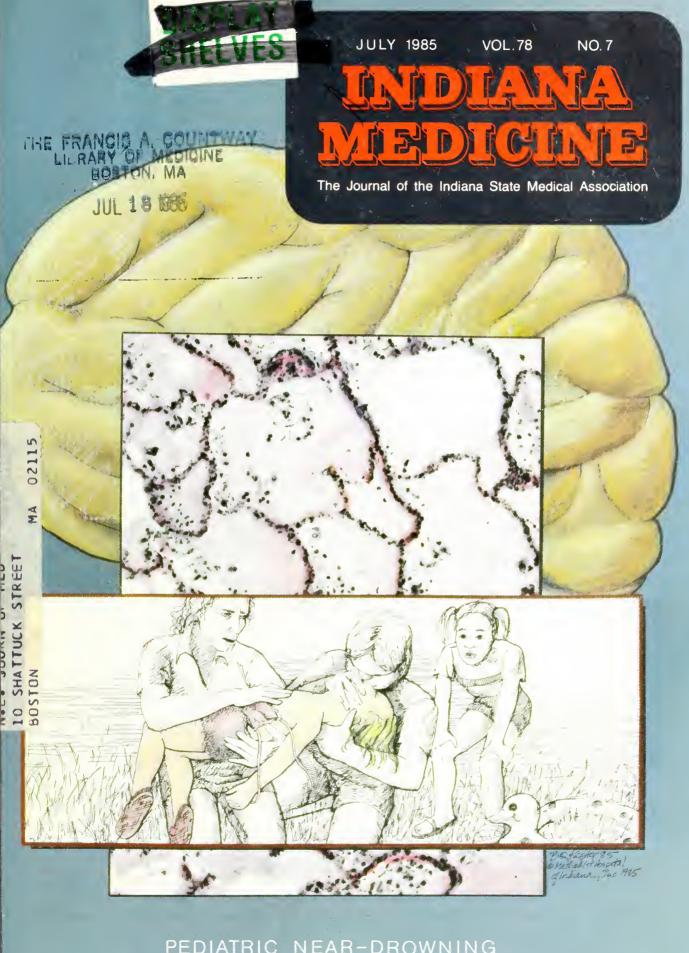
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Vol. 78, No. 7 JULY 1985

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ABOUT THE COVER



Our cover depicts CPR being administered to a child who has nearly drowned. Drowning is a leading cause of death during childhood, and near-drowning is associated with significant morbidity and mortality. Dr. Stephen K. Nugent considers "Pediatric Near-Drowning" in this month's Pediatric Critical Care article.—DRAWING BY BRENDA KESTER, MEDICAL MEDIA PRODUCTIONS, METHODIST HOSPITAL OF INDIANA

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MEDICAL MUSEUM NOTES

CHARLES A. BONSETT, M.D., Indianapolis

R. FRANK B. WYNN (1860-1922) has been mentioned before on this page, most recently as one of three Indiana physicians for whom a mountain has been named (*J Indiana State Med Assoc*, 76:101, 1983). The following letter, dated May 12, 1921, is from Dr. Wynn to William Niles Wishard Jr., who was then a student, inviting young Wishard to join the group of mountain climbers in Glacier (National) Park, Montana, that summer.

Dr. William Niles Wishard Jr. (1898-1973) received the M.D. degree from Harvard Medical School in 1925. Dr. Wishard was an enthusiastic supporter of the museum. This interesting letter is among the items he donated. It reveals much about Dr. Wynn and his mountain climbing activity in Glacier Park:

"Dear doctor Wishard:

"I am enclosing you the tentative itinerary for our trip in the mountains of Glacier Park. This may be modified somewhat as we go along, but in the main it will be adhered to. This round will insure to one who has not previously been there a visit to the most seenic areas, and besides many places off the beaten trail.

"The only condition which would be placed upon William Niles would be that he join the Indiana Nature Study Club, since this expedition is one of a series the Club is conducting in response to the suggestion of Mr. Stephen Mather, Director of National Parks, who is desirous that our organization should take over the charting of the mountain peaks of surpassing interest, climb them, place official records upon the summits, take pictures, and finally submit the routes of ascent to the National Government. Has promised he would print this for us if properly whipped into shape. He is very anxious a Mountain Climbing Manual should be developed, giving routes for hikes and climbs, the purpose being to encour age mountain climbing and hiking as a sport. So our expedition there is



Montage includes Dr. Wynn's invitation to Dr. Wishard to join Indiana's Nature Study Club, which was then planning a trip to the mountains of Glacier Park.

both for the joy of it and with a constructive purpose in mind. We earry to the noteworthy summits a galvanized iron box, containing an Official Record of the Club. We dedicate quite formally every noteworthy summit we ascend, sign the Record, place it in the middle of a cairn, for others to sign should they make the ascent. The Rocky Mountain Club of Denver, the Mazama Mountain Club of Portland are doing a similar work for their respective regions.

"We will aim to follow the harder climbs by a day of rest, when we may fish a little, or take some of the easier or usual hikes.

"Camp Fires are an interesting feature of our outing. These have often been very delightful. There are always a few congenial souls from the outside who ask to join us in these open-air fests, when we sing college songs, have recitations, fun, mock trials, (a la Wishard at Yellowstone) and always some serious talk on a scientific or open air subject.

"Our Party.

"The personnel of our mountain party this year is as follows: "John F. Habbe . . .

"Harry R.W. Horn—Defiance, Ohio—a prince of comrades, who three years ago followed directions we left on the Camp Register at Sun Camp, and climbed Going-to-the Sun Mountain according to those directions, finding the Record, and writing me a letter asking that he might become a member of our next expedition...

"James A. Wynn-for several years a regular participant, but now deprived by professional duties at the Peter Bent Hospital in Boston.

"George Batchelor . . .

"Russell McFall Jr. . . .

"Harry Glossbrenner ...

"F.B. Wynn, otherwise known to the Black Feet Indiana Tribe as Ininapiksi—'Flying Bird.'

"From perusal of this it will be noted that the Presbyterians have an over-weening influence, but the Methodists will do all they can to hold them down to earth."

Sincerely yours

(signed) Frank B. Wynn

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WHAT'S NEW?

Abbott Laboratories has begun worldwide distribution of the first commercial diagnostic tests that employ recombinant DNA (r DNA) technology. The FDA has recently approved two r-DNA products—Corzyme, an enzyme immunoassay (EIA), and Corab, a radio immunoassay (RIA), both of which are for the detection of antibodies to the hepatitis B virus.

AQUA-FLO, Inc., announces a new water purification system. It is applicable to the entire domestic house supply. It softens hard water without adding sodium. It removes particulates such as asbestos fibers. It also removes chlorine and over a hundred toxic waste pollutants. No chemicals are involved in its operation.

Abbott Laboratories announces FDA approval for a new monoclonal antibody test for detecting hepatitis B. Auszyme Monoclonal is the first onestep surface antigen test with monoclonal antibodies. One incubation step is eliminated in the laboratory. This produces faster test results and reduces labor costs by 20%.

Medical Dispensing Systems has developed a new patented pharmaceut ical container, designed for dispensing pre packaged prescriptions. It features child-resistant closures and tamper-evident seals. Labeling is personalized for the doctor. The shape of the containers allows maximum use of space.



"Harold, why do you talk mush here and medicine when we're having a date?"

News of what is new in the medical supply industry is composed of abstracts from news releases by book publishers and manufacturers of pharmaceuticals, clinical laboratory supplies, instruments and surgical appliances. Each item is published as news and does not neces sarily constitute an endorsement of a product or recommendation for its use by Indiana Medicine or by the Indiana State Medical Association.

The Olympus Corporation is offering a 35 mm Polaroid slide outfit. Photomicrography may be accomplished with a new 35mm Polaroid color slide film. Dealers, through October 1985, will offer a complete Polaroid Autoprocess Kit with each Olympus PM-10 35mm camera or AH-2 series microscope which includes a built-in 35mm camera.

A large, randomized clinical trial has shown that a new cisplatin-combination drug therapy has been found to cause fewer side effects than the previously recommended cisplatin-combination. The new combination eliminates some of the unpleasant side effects while achieving the same results.

Haemophilus influenzae type b (Hib), designed to protect young children against serious and potentially lifethreatening infections from the bacterium Haemophilus influenzae type b, is now available. The new vaccine, "b-CapsaTM I" (Haemophilus b polysaccharide vaccine) is manufactured by Praxis Biologics and will be distributed exclusively by Mead Johnson Nutritional Division, a subsidiary of the Bristol-Myers Company.

Williams & Wilkins announces a new text, Current Management of Complications in Orthopaedics: The Hand and Wrist. It was written by 29 expert contributors and was edited by Sigurd C. Sandzen Jr., M.D., Professor of Surgery, Southwestern University, University of Texas Health Science Center. 434 pages, 718 illustrations, \$68.

Searle announces revised and simplified dosing recommendations for Theo-24". FDA-approved new labeling is the result of new data examining the effect of food on absorption of Theo-24. Only patients taking 900 mg or more per day should be instructed to take their dosage at least one hour before eating a high-fat meal.

Hospital Marketing Services has a new skin marking pen. HMS Skin Skribes are used more than any other marking pen. It has a tip at both ends, one for coarse marking and the other for ultra fine marks. They are sold in either sterile or non-sterile condition.

Mead Johnson announces that Desyrel* (trazodone hydrochloride), a widely marketed antidepressant, is now available in a unique Dividose* tablet design. Four common dosage strengths—50 mg, 75mg, 100mg and 150mg—are offered in one easy-to-use tablet. This allows dosing flexibility and assures better patient compliance.

American Medical Systems is introducing a new penile prosthesis which implants with reduced operating room time. The AMS HydroflexTM device uses hydraulic valve technology to permit patients to achieve erections and flaccidity. It is delivered prefilled, which saves time intraoperatively and provides consistent results.

Beckman Instruments has a new booklet on lipemia. The 14-page booklet discusses the laboratory problems which arise with lipemia (cloudy serum). Causes of lipemic serum, a list of 30 specific blood chemical analyses reported to be affected by lipemia and a description of centrifugation to clarify serum are discussed.

Medical Engineering Corporation offers an inflatable penile prosthesis with several improvements over previous methods. Flexi-FlateTM is the first self-contained prosthesis which can be implanted completely within the penis.

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FUTURE FILE

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July 23-25 - Family Practice Update - Part II, Indianapolis.

Aug. 14—Infectious Disease Update, Indianapolis.

Aug. 24-25 – Advanced Trauma Life Support, Indianapolis.

Date Negotiable—Mini-Fellowship in Rheumatology. (For details, see INDIANA MEDICINE, p. 448, June 1985.) For the Specialist

July $8-17-\mathrm{Anatomy}$ & Histopathology of the Head & Neck and Temporal Bone, Indianapolis.

For more information, contact the CME Division, I.U. School of Medicine – (317) 264-8353.

Fire Safety Seminars

The National Fire Protection Association will conduct a series of new Life Safety Code seminars this summer and fall.

One such seminar will meet in Indianapolis Aug. 12 to 15. It is designed for architects, engineers, building inspectors, health care safety personnel and fire officials—and for those whose responsibilities include fire and life safety.

For details, contact NFPA Seminar Registrar, Batterymarch Park, Quincy, Mass. 02269—(617) 770-3000.

"Miss Jones, take off my clothes!"

The Journal of the American Medical Association publishes a list of CME courses for the United States twice yearly. The January listing features courses offered from March through August; the July listing features courses offered from September through February.

Hawaii in March

"Counseling Strategies for Physicians" is the subject of a CME program to be conducted March 24-29, 1986, on the island of Oahu in Hawaii.

Registration fee is \$325. Category 1 credit is 17 hours. The program is being sponsored by the University of Texas. Faculty will discuss social change, family violence, addiction and human sexuality as considerations in dealing with patients; they will present problems likely to be encountered with specific groups of people such as teenagers, minority groups, etc.

For more information call 1-800-332-8747.

Methodist Hospital CME

Sept. 6: Symposium on Current Management of the Failing Heart, Holiday Inn Airport, Indianapolis. (Six hours credit, AMA Category 1).

Sept. 7: 5th Annual House Staff Alumni Meeting, Holiday Inn North, Indianapolis—"Revolution in Medicine: What Lies Ahead."

Sept. 11: Diagnostic Applications of Magnetic Resonance Imaging, Methodist Hospital, Indianapolis.

Sept. 18: Diagnosis and Treatment of Type II Diabetes, Methodist Hospital, Indianapolis.

Sept. 26-27: 5th Annual Harold G. Ochsner, M.D., Radiology Lecture-ship—Bernard Kressel, M.D., guest speaker.

For more information, contact Dixie Mattingly, CME coordinator, Graduate Medical Center, Methodist Hospital of Indiana – (317) 929-3733.

Medical Informatics

The Fifth World Congress on Medical Informatics (MEDINFO 86) will meet in Washington, D.C., Oct. 26-30, 1986.

Applications of those who desire to present papers are now being accepted. Papers are due no later than Jan. 15, 1986.

For further information, contact MEDINFO 86 Organizing Committee, George Washington University Medical Center, Office of CME, 2300 KSt., N.W., Washington, D.C. 20037.

Nutrition and Cancer

"Frontiers of Nutrition and Cancer" will be the subject of a CME program to be conducted Oct. 17 and 18 by the University of Wisconsin and the Wisconsin Nutrition Council. The program, consisting of lectures and panel discussions, will be held at the Holiday Inn Southeast in Madison, Wisc.

Contact Sarah Aslakson, 465B WARF Bldg., 610 Walnut St., Madison, Wisc. 53705—(608) 263-2856.

Pediatric Dermatology

The 13th annual Pediatric Dermatology Seminar will convene at the Eden Roc Hotel, Miami Beach, Feb. 20-23, 1986. The seminar fee is \$240.

Guest speakers will include Raymond Caputo, Andre Nahmias, Sheldon Pollack, Guinter Kahn, Murray Feingold, Kenneth Greer and Christopher Vickers.

For more information, contact Guinter Kahn, M.D., 16800 NW 2nd Ave., Suite 401, North Miami Beach, Fla. 33169 – (305) 652-8600.

Clinical Nutrition

The American Society for Parenteral and Enteral Nutrition will conduct its 1985 postgraduate course, "Perspectives on Clinical Nutrition," Sept. 11 and 12 at the Cathedral Hill Hotel in San Francisco.

For information contact A.S.P.E.N., 8605 Cameron St., Suite 500, Silver Spring, Md. 20910—(301) 587-6315.

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CANCER CORNER

WHLLIAM M. DUGAN, JR., M.D. Clinical Oncology Center, Methodist Hospital of Indiana

4TH NATIONAL SEMINAR ON COMMUNITY CANCER CARE will be held on October 17-20, 1985 at the Hyatt Regency, Indianapolis, Indiana. Proposed curriculum topics are "Ethical Issues in Cancer Care", "Economical Issues in Cancer Care", "The Economics of Outreach Programs", "Screening Programs", "Human Side of Humanomics", "Clinical Research", "Life After Data", "Co-Ventures—Is This A Viable Alternative", and "Marketing: Striking the Balance Between Winning and Losing".

Mini-Session Topics are "Patient/Family Education Programs", "Screening Programs", "Hospice—Current Issues", "Volunteer", "Patient/Family Needs", and "Outreach Programs".

For further information, please call (317) 929-3733, the Office of Continuing Medical Education, Methodist Hospital of Indiana, Inc.

LEUKEMIA RESEARCH GRANT APPLICATIONS: The Leukemia Society of America is now accepting applications for 1986 grants to encourage research at both the basic science and clinical levels in the fields of leukemia and related diseases.

According to Edwin Ades, Ph.D., chairman of the Indiana Leukemia Society's Professional Education Committee, the awards are a primary source of salary support for individuals whose work is concentrated on seeking the causes and eventual cures for leukemia, the lymphomas, Hodgkin's disease and multiple myeloma.

The Society offers three awards: 1) Five-year Scholar grants for a total of \$150,000 to researchers who have demonstrated over a period of not less than five years, their abilities to conduct original investigations in the specified fields; 2) Three-year Special Fellow grants for a total of \$63,000 for those investigators in the intermediate stages of career development; 3) Two year Fellow grants for a total of \$34,000 for promising investigators with no or minimal prior

experience assisting and training with scientists and physicians in the related fields. In all categories, candidates should hold a doctoral degree but may not have attained tenure status.

Deadline for filing applications with the Society is September 1, 1985. Only one application in each grant category from any one department or faculty sponsor may be submitted. Proposals will be evaluated on a competitive basis by the Leukemia Society's Grant Review Subcommittee in January, 1986 with funding to start July 1, 1986. For application forms and additional information, write to Research Grant Coordinator, Leukemia Society of America, 733 3rd Ave., New York, New York, 10017.

CLINICAL TRIALS: The National Cancer Institute (NCI) is pleased to introduce a new publication, What Are Trials All About?, for patients who are considering taking part in trials for cancer treatment. The purpose of the NCI booklet is to explain the nature of clinical trials in lay language and to supplement the information patients receive from health professionals. The booklet presents basic information about clinical research in a simple question-and-answer format.

If you would like to order copies of this booklet, mail your request to: Office of Cancer Communications, National Cancer Institute, Building 31, Room 10A18, Bethesda, MD 20205 or call toll-free, the Cancer Information Service at 1-800-4-CANCER.

1985 KILMER PRIZE: Purdue University pharmacy student, Mark T. Gill of Seymour, is the winner of the 1985 Kilmer Prize for research in natural medicinal products. The nation-wide competition, administered by the Academy of Pharmaceutical Sciences of the American Pharmaceutical Association and the American Society of Pharmacognosy, is open to both graduate and undergraduate students doing research in pharmacog-

nosy, which deals with natural drugs.

Gill's research centered on the inhibition of crown-gall tumors on discs of potato tubers and on the use of brine-shrimp toxicity to produce two new antitumor compounds. Pharmacognosy Professor Jerry L. Mc-Laughlin explained that if a drug inhibits crown-gall tumors in plants, it will probably have antitumor activity in animals.

The Purdue study, conducted in the Department of Medicinal Chemistry and Pharmacognosy laboratories under McLaughlin and postdoctoral research associate Renu Bajaj, is part of a project sponsored by the NCI and aimed at discovery of new anticancer agents through simple, animal-sparing analyses. Gill's achievement also marked the third straight year that an undergraduate in McLaughlin's laboratory won the Kilmer Prize.

NCI DIET, Nutrition & Cancer 1983 status report is available free from Capital Systems Group, phone 301-881-9400. The report summarizes nutrition related research supported by NCI and describes future research directions.

"DECADE OF PROGRESS": The 1984 Cancer Care Annual Report, "Decade of Progress" highlights ten years of quality cancer care at Deaconess Hospital, Inc., Evansville, Indiana. The report outlines numerous accomplishments during the 10 years such as repeated accreditation for the Tumor Registry from the ACoS, completion of a 4 year NCI contract to organize model community cancer care programs and state-of-the-art technology. High quality cancer care with emphasis on education and early detection has resulted in improvements in survival rates. For example, colo-rectal survival rates improved approximately 20% during the last 10 years. To receive your FREE copy of the 1984 Cancer Care Annual Report, pelase call the Office of Oncology at (812) 426-3602.



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PUBLIC HEALTH NOTES

Industrial Waste Disposal Study Shows High PCB Levels at Monroe County Sites

In late 1983, the Indiana State Board of Health (ISBH) asked the Centers for Disease Control (CDC) for assistance in studying exposure to and health effects from PCBs (polychlorinated biphenyls) among residents near three Monroe waste sites.

After consultations, ISBH and CDC developed a pilot study designed to accomplish several purposes:

- to evaluate persons at high risk of exposure to these waste sites and determine if any of these individuals have abnormally elevated serum PCB levels:
- · to determine which environmental pathways containing PCBs might have contributed most to producing abnormally elevated levels of PCBs in human sera; and
- to compare health parameters with serum PCB levels.

Bloomington, a city of approximately 53,000, has a long and detailed history of industrial waste disposal compounded by unusual geologic features that make the environment susceptible to pollution by such wastes.

In 1976, PCBs were found in the influent [entering] and effluent [leavingl wastewater of the sewage treatment plant in Bloomington, and in sewage sludge obtained from the plant by citizens who used it for fertilizer in their gardens.

A study done at that time showed workers in electrical equipment manufacturing, members of these workers' families, and sewage treatment plant workers had elevated serum PCB levels compared to either sludgeusers or a population control group. Other possible sources of community exposure included the common practice of scavenging metal from capacitors that did not meet quality

TABLE

Geometric Mean Total Serum PCB Levels by Risk Group Strata Bloomington, Indiana-1984

Risk Group	Serum PCB (parts Geometric			s per billion, ppb) % Greater than	
Stratum	N	Mean	Range	or Equal to 20 ppb	
Total HIGHEST RISK Group	61	10.91	3.0-75.0	19.7	
Occupational exposure	10	27.66*	15.0-75.0	70.0	
Scavenging	11	12.18	4.0 - 51.0	18.2	
Swimming	-6	9.03	3.0-23.0	33.3	
Fish-eating	7	8.76	4.0 - 18.0	0.0	
Digging	9	8.67	4.0 - 42.0	11.1	
Playing	-6	7.92	5.0 - 12.0	0.0	
Closest residence	5	7.85	3.0 - 13.0	0.0	
Game-eating	7	7.69	5.0 - 12.0	0.0	
HIGHEST RISK Group					
(excluding workers)	51	9.03	3.0 - 51.0	9.8	
UNEXPOSED Group	8	5.87	4.0-13.0	0.0	
RANDOMLY SELECTED AT RISK Group	55	9.03	2.0-47.0	16.3	

^{*-}Stratum mean significantly different (p < 0.05) from all other stratified means in pair-wise comparisons within the highest risk group.

standards which were discarded at several dump sites, swimming in surface waters on or adjacent to these sites, and consumption of contaminated locally eaught fish. The three dump sites which were the focus of most attention relating to human exposure were Lemon Lane Landfill, Bennett's Stone Quarry, and Neal's Landfill.

Based on results of a screening survey conducted last year of 995 individuals in Monroe County, 114 individuals participated in the study. This group included 61 individuals who were considered to be at highest risk of exposure because of characteristic activity patterns in contaminated areas, eight persons who appeared to have had no exposures, and 45 randomly selected participants from the population at risk.

As shown in the Table, the geo-

metric mean serum PCB level for the highest risk group is greater than that for both the unexposed group and the randomly selected at-risk groups. However, when workers with known occupational exposures to PCBs are removed from the highest risk cohort, statistically significant excess serum PCB levels in this group versus either of the two groups disappears.

Previous studies of nonoccupationally exposed, randomly selected populations in areas of the United States have demonstrated geometric mean serum PCB levels of approximately six parts per billion. In addition, these studies have shown that 95% of the tested individuals should show serum PCB levels at levels less than 20 parts per billion. Both the highest risk group and the randomly selected at-

CONTINUED ON PAGE 608



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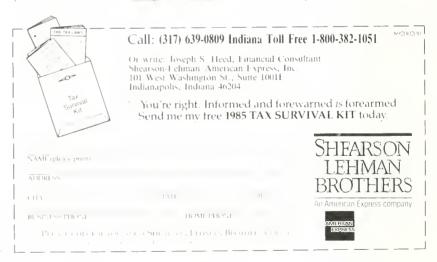
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As an organization accredited for continuing medical education, the Indiana University School of Medicine certifies that this CME activity meets the criteria for one credit hour in Category 1 for the Physician's Recognition Award of the American Medical Association, provided it is used and completed as designated.

To obtain Category 1 credit for this month's article, complete the quiz on page 623.



Prolonged Apnea in Infancy: Evaluation and Management

PETER H. SCOTT, M.D. Indianapolis

From the Section of Pulmonology, Dept. of Pediatrics, Indiana University School of Medicine, and the Children's Apnea Center, James Whitcomb Riley Hospital for Children, Indianapolis.

Reprints: Peter H. Scott, M.D., James Whitcomb Riley Hospital for Children, Rm. 293, 702 Barnhill Drive, Indianapolis, Ind. 46223.

LINICALLY SIGNIFICANT APNEA occurs in patients of all ages but is prevalent especially in neonates and infants. Although the incidence of apnea in infants is unknown, large groups of newborn and infant patients, who represent collectively thousands of children born annually in Indiana, are at increased risk for apnea. High-risk patient groups include premature infants, infants with serious acute infections, neonates and older infants with seizure disorders, subsequent siblings of sudden infant death syndrome (SIDS) victims, and children with congenital or acquired anomalies of the upper respiratory tract. Additionally, the growing awareness of SIDS and its possible relationship to ventilatory dysfunction make a review of infantile apnea timely.

Normal Breathing Pauses

Apnea is a breathing pause of any duration. All infants have brief apnea pauses during wakefulness and sleep. Observant parents may report breathing pauses of 5 to 15 seconds which often occur after a yawn or sigh. Another common breathing pattern in neonates and infants is periodic apnea, a series of short apnea pauses interrupted at regular intervals by normal respirations. Periodic apnea is seen during up to 3% of sleep time in normal infants. Brief apnea pauses also can be seen during regurgitation of gastric contents. These episodes occur when laryngeal chemoreceptors are stimulated and are often accompanied by plethora. These episodes should be considered normal unless they occur frequently or are accompanied by more significant symptoms, such as transient loss of consciousness or cyanosis. It is important that the clinician know when to be concerned about an episode reported by parents and when he can reassure them that the apnea was not significant.

Prolonged Apnea

In contrast to the short apnea pauses seen in most normal infants, prolonged apnea is a clinically significant breathing pause and is best viewed as a symptom or sign. Prolonged apnea is characterized by an apnea pause of 20 seconds or longer, or a shorter episode of apnea associated with cyanosis, pallor or bradycardia.

Prolonged apnea may be central or obstructive in nature. In central apnea, chest and abdominal wall motion, and thus air entry, cease. Central apnea occurs usually during sleep and may be accompanied by cyanosis or pallor.

Obstructive apnea occurs when the nasal, oropharyngeal or laryngeal air way becomes obstructed, and despite vigorous ventilatory muscle contraction, air entry cannot occur. Obstructive apnea can occur during sleep or wakefulness. During an episode of complete obstruction, cyanosis and bradycardia often develop within 10 to 15 seconds. Some infants have a mixed pattern that includes central and obstructive apneas.

Differential Diagnosis

Prolonged apnea can result from many acute or chronic conditions (*Table 1*). Some are seen more frequently in certain age groups. For example, whereas foreign body aspiration is encountered typically in the older infant, a diagnosis of idiopathic congenital hypoventilation is made only in the neonatal period.

Signs that accompany prolonged apnea frequently can help narrow the diagnostic possibilities. Prolonged apnea associated with vomiting after feedings is frequently secondary to

TABLE 1 Differential Diagnosis of Prolonged Appea in Infants

Central Apnea Infectious	– Bacterial sepsis; menin	Pulmonary	Pneumonia Bronchiolitis	
	gitis;	Obstructive Apnea		
	pneumonia; pertussis; infantile botulism —Viral respiratory syncytial	Nasul	-Choanal stenosis Excessive nasal secretions Adenoid hypertrophy	
	virus adenovirus	Oropharyngeal	 Micrognathia Tonsil hyper- trophy 	
Metabolic	– Acidosis Hypoglycemia Hyponatremia	Laryngeal	– Laryngoma- lacia	
CNS	- Seizure disorder Meningitis Hydrocephalus Idiopathic con-		Web/Polyp Gastroesopha- geal reflux Arnold-Chiari malforma- tion	
	genital hypoventila- tion Leigh's syndrome	Tracheal	— Tracheoma- lacia Tracheal stenosis Vascular ring	
Cardiac	-Congestive heart failure		Foreign body aspiration	

gastroesophageal reflux. Prolonged apnea and cough may be due to pertussis, bronchiolitis, or foreign body aspiration. In young infants with meningitis specific signs of meningeal irritation, such as nuchal rigidity, usually are lacking. However, meningitis is a likely diagnosis in the child with prolonged apnea and either lethargy or extreme irritability. Prolonged apnea may be the initial symptom in all of these conditions, and at the time of presentation, few diagnoses may be excluded entirely.

Evaluation

All patients with suspected prolonged apnea should be evaluated in the hospital. Hospital admission serves several purposes. Continuous cardiopulmonary monitoring can be accomplished easily, and laboratory studies can be expeditiously obtained. Parents are much less anxious if they know their child is being monitored by trained health professionals. However, evaluation on a hospital ward has limitations. The noise level of an in-patient unit far exceeds that of the patient's home. Thus, sleep patterns can be altered dramatically. and prolonged apnea may not occur. No parent should be accused of fabrication simply because the reported prolonged apnea cannot be documented in the hospital.

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History: Meticulous attention to obtaining an accurate history is mandatory. Coupled with a thorough physical examination, the history is the only means by which the physician can decide that the episode represented a normal breathing pattern or that further evaluation is necessary. The frequency of episodes and their association with other processes such as feeding or crying are important clues as to which diagnostic entities to consider. Parents, who are naturally anxious at the time of the apnea, often are unable to report accurately the duration of the initial episode. They can report frequently whether the child was asleep at the onset of the episode and whether a change of skin color occurred.

A significant color change includes pallor or cyanosis. These should not be confused with plethora, a flushing that often accompanies choking or prolonged crying. Any child with pallor or cyanosis during a breathing pause, regardless of its duration, has prolonged apnea and should be evaluated.

The state of consciousness of the child at the onset of the episodes is important to elicit. Prolonged apnea during wakefulness usually is less severe than sleep apnea, and an underlying cause of apnea is more likely to be identified in patients who have episodes during wakefulness. For example, the child with prolonged apnea secondary to gastroesophageal reflux has episodes typically after a feeding, while he is still awake.

The presence of flaccidity or increased muscle tone during the episode suggests a seizure disorder as the diagnosis. Unusual eye movements may also indicate seizure activity. In the older infant or young child, a history of persistent snoring during sleep may alert the clinician to the presence of partial airway obstruction. These patients may have episodes of no apparent ventilatory effort followed by gasping and re-

sumption of quiet breathing or snoring.

Occasionally, a family history of sudden infant death syndrome or sleep apnea (in adults or children) can be obtained.

Physical Examination: Prolonged apnea can be the first sign of a serious, acute illness. A thorough physical examination is essential to rule out an acute process (e.g., meningitis) as well as a significant chronic condition (e.g., seizure disorder).

A detailed examination of the nasal and oral airways should be performed. The presence of a cleft palate, stenotic choanae or micrognathia may serve as a clue to the specific diagnosis.

Airway obstruction is difficult to detect in infants. Snoring occurs infrequently. However, paradoxical movement of the chest and abdominal wall suggests partial or complete extra-thoracic airway obstruction.

Particular attention should be paid to the ventilatory pattern during wakefulness and sleep. Observing the child during sleep may unmask certain conditions not suspected while examining the alert child. Additionally, the child should be observed in different positions; the oropharyngeal airway may be patent with the patient in the prone position but nearly totally occluded when he is supine.

Laboratory Evaluation: Patients should be evaluated as soon as possible after the initial event. Even in the child who has apparently returned to a normal state of consciousness, a low serum bicarbonate value secondary to acidemia may indicate that he has sustained an episode of severe hypoxia. A normal serum glucose, lactate or ammonia, especially if obtained at the time of the episode, may be helpful in excluding significant metabolic disease. A hematocrit should be performed to rule out anemia; an elevated white cell count may be indicative of an infectious process.

Suspected cardiopulmonary disease can be further investigated with a chest radiograph and electrocardiogram. Echocardiography also may be helpful if cardiac disease is likely. If an arrhythmia is the suspected cause of the episode, a 24-hour electrocardiographic recording may be indicated even if the initial electrocardiogram is normal.

Electroencephalography should be performed in any infant with a suspected seizure disorder. However, a normal electrocephalogram (EEG) does not exclude entirely this diagnosis. In some patients, prolonged EEG recordings obtained during sleep may be helpful. In patients with multiple anomalies or with an abnormal neurologic examination, a computed tomographic scan of the head may be indicated.

In patients with a history suggestive of gastroesophageal reflux or aspiration during feeding, the laboratory evaluation should begin with a contrast radiographic study of the upper gastrointestinal tract. Nasopharyngeal incompetence, swallowing dysfunction with aspiration and esophageal dysmotility may be observed in addition to gastroesophageal reflux. Conditions causing partial gastric outlet or proximal intestinal obstruction may predispose infants to gastroesophageal reflux. Thus, the examination should not be considered complete until the barium reaches the ligament of Treitz. A normal contrast radiographic examination does not exclude gastroesophageal reflux, which may be demonstrable with other diagnostic studies including gastroesophageal scintiscan, esophageal manometry or esophageal pH monitoring.

Ventilatory patterns during sleep may be recorded using two different techniques. Pneumocardiogram recordings may be obtained in the hospital setting or at home. These 12- or 24-hour patterns of chest wall motion and heart rate are recorded either on magnetic tape or directly onto recording paper. Analysis of these data vields information about a child's ven tilatory pattern and heart rate at the time of the test. A finding of prolonged apnea is clearly abnormal and indicates that risk for future significant apnea is increased. The interpretation of an increased number of short apnea episodes (6 to 20 seconds) or increased periodic apnea is less clear. In patients who have increased short or periodic apnea and whose history suggests that prolonged apnea is likely to recur, a respiratory stimulant such as theophylline may be indicated to normalize the ventilatory pattern.

The pneumocardiogram is not useful in detecting obstructive apnea, as it does not allow for monitoring gas flow at the nose and mouth. The pneumocardiogram is not a screening test to detect infants at risk for sudden infant death syndrome.

More accurate ventilatory measurements during sleep can be obtained using sophisticated monitoring equipment in conjunction with a multichannel recorder (polysomnogram). Sleep staging can be performed using EEG and eye electrodes. Nasal and oral gas flow can be detected with thermistors, and quantitative lung volume measurements can be made using inductive plethysmographic methods. Ventilation can be assessed by a carbon dioxide analyzer, which measures carbon dioxide tensions of end-expiratory gas. A transcutaneous PO. monitor or an ear oximeter is used to monitor oxygenation.

As with any laboratory investigation, management decisions should not be based solely on pneumocardiogram and polysomnogram data. Although these studies serve as useful guides to therapy, some patients with severe recurrent apnea have repeatedly normal polysomnograms.

Apnea of Infancy

In more than half of the patients evaluated for prolonged apnea, no specific underlying cause of the child's episodes can be found. An infant with a history of prolonged apnea that is unexplained after an appropriate in vestigation has apnea of infancy. Sometimes referred to as "near-miss," "aborted" or "interrupted SIDS", the term "apnea of infancy" is preferred for several reasons. Although patients with apnea of infancy are at increased risk for SIDS, the term "near-miss SIDS" implies the child would have died of SIDS had intervention not been prompt; this can never be proved. In addition, anxious parents are not served well by hearing that their child "nearly died" each time their child's diagnosis is mentioned. "Apnea of infancy" is descriptive, properly avoids an unproved association with SIDS, and is preferred to other proposed terminol-

Most patients with apnea of infancy have subsequent prolonged apnea after the initial evaluation. However, most episodes are associated with no other significant symptoms. In about 90% of children who require a home apnea/bradycardia monitor for apnea of infancy, the monitor can be discontinued between 6 and 12 months of age.

Management

If prolonged apnea is due to a demonstrable underlying cause (e.g., seizure disorder), the condition should be treated. The course of treatment of some patients, such as those with meningitis, may need to be completed before discharge. Even for patients with potentially chronic conditions (e.g., gastroesophageal reflux) therapy may need to be initiated while the child remains in the hospital, until the response to treatment can be defined.

Patients with apnea of infancy are managed primarily with apnea/bra-dyeardia monitors designed for use in the home. In some patients at risk for frequent or severe prolonged apnea, the addition of a methylxanthine may be necessary.

Home Monitoring: Apnea/bradyeardia monitors are not a treatment in that they do not prevent prolonged apnea or bradycardia. Monitors are designed to alert parents to a potentially life-threatening episode. Although monitors are now used widely, several questions remain about their utility. It is unclear whether monitors prevent deaths due to SIDS; indeed, children with apnea of infancy have died of SIDS despite appropriate monitoring and resuscitation efforts. Some physicians feel monitors impose additional emotional burdens on families already under stress. The cost of monitoring is substantial and adds to these burdens.

However, studies have shown that home monitors relieve, rather than add to, much of the anxiety many parents must deal with. The use of monitors in the home has allowed for earlier hospital discharge than would otherwise be feasible for patients with persistent apnea or bradycardia. For these patients early discharge may save thousands of health care dollars. Thus, despite some concerns with the growing use of home monitors, apnea of infancy is a clinical problem of increasing significance, and apnea/ bradycardia monitors are judged by most apnea center directors as the initial step in managing these patients.

Once a decision to initiate home monitoring has been made, training of the parents and other caretakers, (e.g., extended family members, babysitters) begins. The most appropriate setting for instruction is an inpatient unit of the hospital. Since there are too many details of monitoring and resuscitation techniques to cover during a single session, we do not instruct patients' families at home or in the clinic.

A multi-disciplinary approach to education works well. Our Center's family education protocol is outlined in *Table 2.* Significant overlap of responsibilities has advantages. For example, parents may hear about similar

aspects of monitoring from the nurse and the respiratory therapist involved in instruction. This reinforcement serves to emphasize a uniform approach to monitoring that is important when multiple caretakers are involved.

Several acceptable monitors are available. All of these are based on impedance pneumography. Magnet/pad apnea monitors are associated with many false alarms, have no provision for monitoring heart rate and are not suitable for home use.

Initially, an apnea time delay of 15 seconds is chosen. If no apnea alarms associated with color changes or bra-

TABLE 2 Family Education Protocol

Apnea Center Nurse
Assess home environment
Electrical power and telephone services
Need for remote alarm
Bedside supplies (alarm log, flashlight, etc.)
Arrange instruction for extended family, babysitters
Electrode placement; skin care

Public health referral (if neces-

Supplier of Monitor Initial monitor instruction

sary)

Respiratory Therapist
Reinforce proper use of monitor
Appropriate response to alarms
CPR instruction

Social Worker
Assess adjustment to home monitoring
Financial assessment and counseling
Support group referral
Home monitor support group
Local SIDS Foundation
chapter
(only if previous SIDS death in family)

dycardia oceur within one month, the apnea delay is adjusted to 20 seconds. In infants less than 2 months of age (corrected for gestational age) the bradycardia alarm limit is set typically at 80 beats per minute. This is lowered to 70 and 60 beats per minute at 4 and 6 months of age, respectively. These should be viewed only as guidelines. Oceasionally, an infant will have frequent but transient bradycardia alarms that are not associated with a color change or the need for stimulation. In these infants it is usually safe to lower the alarm limit below the value outlined above. If there is a question about the nature of the episodes, a pneumocardiogram done in the home may be helpful in defining the extent and frequency of bradycardia.

Before discharge all caretakers of the infant must feel comfortable with monitoring and resuscitation techniques. In addition, many parents are intimidated initially by the monitor and unsure of their new monitoring skills. These feelings, added to parents' concern for their child, require a compassionate approach. Input by nurses, therapists and social workers knowledgeable about monitoring techniques and pitfalls is an essential component of the education of these families.

Criteria that must be met before a monitor is discontinued are shown in Table 3. We do not consider discontinuing a monitor until the child is at least 6 months of age. Until that time parents are advised to monitor children during all periods of sleep, including naps. Once the criteria to discontinue monitoring have been met, there is no need to gradually "wean" the patient from the monitor.

Many parents report an increase in the frequency of apnea alarms after a DPT immunization. These episodes rarely are severe. Thus, there is no need to withhold DPT immunization in patients with apnea of infancy, and, unless otherwise contraindicated, these patients should receive routine TABLE 3 Criteria for Discontinuing Monitor

Within previous 2 months:

No apnea alarm (20 second apnea delay)

No bradycardia alarm requiring intervention
(stimulation or resuscitation)

No use of methylxanthine

Normal pneumocardiogram

A viral illness or immunization

Within previous 3 months:

No apnea alarm requiring intervention

childhood immunizations.

Theophylline: Unlike apnea/brady-cardia monitors, theophylline is a treatment for disorders of ventilatory control. Theophylline and related methylxanthines, like caffeine, reduce the frequency and duration of short apneas and periodic apnea episodes.

We consider theophylline for patients who are maintained on a home monitor and who have an abnormal pneumocardiogram or polysomnogram. Theophylline is also used in patients with a normal study who have severe, recurrent apnea. Methylxanthines generally should be avoided in patients with gastroesophageal reflux and in those with seizures, as these drugs can exacerbate these conditions.

The dose requirement of theophylline is much less in infants than in older children. In neonates the starting dose of anhydrous theophylline is 4 mg/kg/24 hours and can be divided every 8 or 12 hours. In the older infant, a starting dose of 8 mg/kg/24 hours divided every 6 or 8 hours may be used.

Theophylline usually controls apnea and bradycardia when serum concentrations are in the 8 to 12 mg/L range. However, some patients who respond poorly to serum concentrations less than 12 mg/L, are controlled well with concentrations

between 15 and 20 mg/L. If serum concentrations are above 20 mg/L, or if vomiting or extreme irritability occur at any concentration, the dose should be reduced.

Summary

We have described the normal and abnormal ventilatory patterns in infants and have outlined our approach to the evaluation and management of patients with prolonged apnea. It is most important to differentiate acute apnea which could be secondary to a

treatable infectious process, from conditions of a more chronic nature. The physician must seek a specific diagnosis and start appropriate therapy in each patient who presents with prolonged apnea. The home apnea/bradycardia monitor is used primarily in the management of patients with apnea of infancy, and theophylline serves as an adjunct. Anticipating problems with families during the initial instruction serves to avoid difficulties during the monitoring course.

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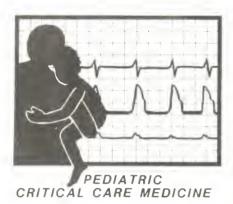
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Pediatric Near-Drowning



ROWNING IS A submersion incident resulting in asphyxia and death while submerged or within 24 hours of the incident. Near-drowning is a submersion incident followed by at least temporary (24 hours)

STEPHEN K. NUGENT, M.D.

Indianapolis

survival regardless of eventual outcome.

Epidemiology/Prevention

Childhood Hazards
That Are Often
Overlooked by Parents
Can Lead to
Preventable Drowning
or Near-Drowning
Incidents . . .

State and national drowning statistics are readily available; near-drowning statistics are not. Drowning victims represent "the tip of the iceberg" relative to near-drowning victims transported to hospitals for definitive care. Approximately 6,000 drownings occur in the United States vearly. From 1980 through 1983, 362 drownings occurred in Indiana: 10<1 year of age, 49 1-4 years, 21 5-9 years, 26 10-14 years, and 56 15-19 years (162 of 362 total). Drowning is a leading cause of death during childhood, and near-drowning is associated with significant morbidity and mortality.

An understanding of drowning and near-drowning epidemiology is crucial as prevention is so often possible. Submersion incidents involving children 1-4 years are most often associated with inadequate adult supervision and/or inadequate pool barriers which don't effectively limit pool access. Toddlers have often not developed an appropriate respect for water and may be tempted by objects in or near water. They may use the patio around a pool as a play area for wheeled toys such as tricycles or toy trucks. Incidents involving older children may occur when a supervising

adult is overeonfident of the child's swimming ability. Those during adolescence may be related to overestimation of swimming skills or overextending one's abilities, showing off, attempts to swim long distances, and with increasing frequency to alcohol and drugs. Head trauma at any age may be associated with a submersion incident. Hyperventilation to prolong underwater swimming is particularly dangerous. Hyperventilation increases the breath-holding breaking point; with the exercise of swimming underwater, hypoxemia occurs before hypercapnia. Unconsciousness from cerebral hypoxia may cause drown-

Bathtub, hot tub, and pail immersion incidents may occur. Bathtub incidents often involve a child less than one year left attended by a sibling less than four years in a highly mobile, large family of low socioeconomic status. The infant can sit alone and pull to stand, leading a parent to believe that the infant can keep his head out of water without problem. Hot tub incidents typically involve an unattended child who falls into an uncovered tub, or a child trapped by the suction of a hot tub without a second safety suction outlet to prevent the formation of a vacuum when one outlet is occluded. Pail immersions involve the unattended child of 9-15 months who can pull to stand on a 3-5 gallon pail and not be able to extricate head and shoulders. Parents typically did not consider the pail of water to be hazardous. Pediatricians and family practitioners have the opportunity to play a major role in the prevention of these incidents.

Though debatable, submersion accidents in children with epilepsy may occur at a rate four times that of the

Dr. Nugent is Medical Director, Pediatric Intensive Care Unit, Methodist Hospital of Indiana, 1604 N. Capitol Ave., Indian apolis, Ind. 46202. normal population. Lack of fully controlled seizures, mental retardation, and occurrence in a bathtub are char acteristic. Orlowski proposed the following guidelines for epileptics, whether mentally normal or subnormal: (1) seizures well controlled with stable therapeutic anti-convulsant levels; (2) swimming only when a lifeguard is informed or a competent swimming companion is present for proper supervision. Bathing instructions for patients with epilepsy include close supervisijon of young children. For older patients, it is recommended that bathtub water depth be 5-7.5 cm. maximum, or to shower while seated on the bathtub floor with drain open or using a hand-held show ering instrument.9

Predictions are that submersion ineidents, especially near-drowning, will increase in number. Population growth, more frequent and aggres sive eardiopulmonary resuscitation (CPR), more private pools and participation in aquatic sports generally, and use of alcohol and drugs in adolescents are commonly cited. Ways of preventing submersion incidents are obvious from the above discussion of epidemiology. Further suggestions include mandatory CPR certification for pool owners, improved community education regarding magnitude of the problem, establishment of definitive water-safety rules within the family, and swimming lessons for children, among others.10 It is difficult to underestimate the role that physicians can play in preventing these incidents.

Management

The most important consequence of near-drowning is hypoxemia, whether from laryngospasm and upper airway obstruction or aspiration of water and often gastric contents." All patients submerged, whether CPR is required or not and regardless of level of consciousness, should receive 100% oxygen. Assessment of oxygenation in the emergency room (ER) by arterial

TABLE

Unfavorable Prognostic Factors

CPR in ER^{(a,y)-1}
Initial pH < 7.0°
Coma after resuscitation (a,y)-1
Coma with fixed and dilated pupils (a,y)
No spontaneous respiration after CPR (Glasgow Coma Score < 5 in ER^(a))
Orlowski score ≥ 3: 5% survival (age < 3 yrs.)
maximum submersion time > 5 minutes
CPR not attempted for at least 10 minutes after rescue coma on admission
arterial pH < 7.10
Asystole (beating heart at ER arrival = 100% intact

Asystole (beating heart at ER arrival = 100% intact survival):

Elevated intracranial pressure 1...29.30.11

blood gas or ear oximetry is the prior ity in the patient who arrives with stable vital signs. If spontaneous ven tilation occurred while submerged, aspiration occurred and alteration in pulmonary function is likely. Pathophysiologic changes include inactivation of pulmonary surfactant, alveolarcapillary membrane permeability abnormalities, reduced lung volume and compliance, ventilation-perfusion mismatch, and hypoxemia. A 24-hour admission should detect pulmonary or neurologic deterioration in the patient who sustained a significant submersion accident but appears to have normal pulmonary and neurologic function on ER arrival.

In every patient who does not immediately respond to CPR, CPR should be continued en route (with neck stabilizing technique) and in the ER. Assessment of neurologic status, correction of inevitable metabolic acidosis, and optimum CPR are the priorities. Hopefully, accurate information can be obtained about duration of submersion, vital signs and clinical state when removed from water, adequacy of CPR since rescue, and body temperature. In this setting, the ER physician may be faced with a difficult decision regarding aggressiveness and duration of CPR. Resuscitation required in the ER has been a consistent indicator of poor outcome for near-drowning patients, especially when neurologic function is absent. Yet enough warm water (>20°C) exceptions have occurred to justify aggressive ER CPR. A recent series described 24% intact survival in 66 pediatric near-drowning victims who required CPR in an ER and had an ER (not ICU) Glasgow Coma Score of 3 (no eye opening, no verbal response, no movement to pain). " How ever, given a knowledge of prognostic factors and the circumstances of the specific episode, I feel the following recommendation is reasonable: given a body temperature >32° (89.5°F), lack of any neurologie function or movement to pain, and inability to generate spontaneous heart rate after 30-40 minutes of effective ER CPR, cessation of CPR efforts may be considered.

A complete discussion of near drowning and cold water immersion can be found elsewhere. It should be emphasized that infants and children are routinely mildly hypothermic after an immersion incident. Core temperature drops more rapidly given a relatively large ratio of surface area to body mass. The cold water diving reflex is especially active in infants and children. This peripheral vasoconstriction with continued cer-

ebral and cardiac blood flow, plus reduced cerebral metabolic demands from extreme hypothermia, are felt responsible for reports of normal survival after prolonged periods of cold water immersion. Body temperature must be assessed in all near-drowning victims. Below a core temperature of 27 C, evidence of life may be difficult to detect, i.e., clinical and biologic death may not be synonymous.13 Rewarming (passive external, active external, active internal) and CPR should be continued simultaneously until a temperature of at least 32°C is reached. The criterion for death then becomes failure to respond to CPR after rewarming to a core temperature of at least 32°C.

Once cardiovascular stability is achieved, intensive care unit (ICU) admission is mandatory. Monitoring appropriate for any critically ill pediatric patient is instituted (heart rate, respiration, noninvasive or invasive blood pressure, and temperature; daily weight and intake and output; arterial blood gas, electrolyte, and ear oximetry oxygen saturation measurements; hourly neurochecks; pulmonary artery catheterization if indicated by cardiovascular and/or pulmonary dysfunction; and infection surveillance). Airway, mechanical ventilation, and respiratory care techniques applicable to the neardrowning victim with pulmonary involvement have been described.14 Steroids are not indicated for central nervous system management and questionably for pulmonary involvement. 15 Antibiotics are generally not indicated, though grossly contaminated water or aspiration of water from a hot tub (given Pseudomonas proliferation and reports of fatal pneumonia and septicemia) should make one more strongly consider their use.

A fatal outcome from near-drowning is most often the result of cerebral hypoxia and ischemia. Basic ICU management of neurologic dysfunction includes adequate oxygenation

and prevention of hypercarbia, maintenance of normal blood pressure, prevention of hypervolemia with mild dehydration desirable, prevention of seizures and hyperthermia, and the use of sedation and neuromuscular paralysis if needed for hyperrigidity or posturing. Vigilance for hyponatre mia from excessive antidiuretic hormone secretion may prevent further cerebral and pulmonary deterioration. Controversy surrounds the question of more aggressive cerebral resuscitation therapy (CRT: intracranial pressure monitoring, barbiturate coma, intentional hypothermia to 30-32°C). Awake, fully conscious patients and those who are obtunded. stuporous but arousable, with purposeful response to pain and normal respiratory pattern, should survive with normal neurologic findings. 17.18 The question is whether CRT improves neurologic outcome in comatose patients who are decorticate, decerebrate, or flaccid at ICU admission (see below).

Outcome

Knowledge of factors related to outcome is helpful in terms of prognosis and mandatory relative to ICU level of support. Intact survival is most related to CPR response at the scene. This response is in turn a function of the duration of hypoxia (as modified by profound hypothermia) and the immediacy and efficacy of CPR. Unfavorable prognostic factors described in the literature are summarized in the Table. Unfortunately, no single factor or group of factors can be identified that totally precludes a complete recovery. Hence, the uniform recommendation that aggressive on-the-scene and ER resuscitation be undertaken regardless of clinical state. Comparison of outcome from one series to the next is difficult because of dissimilarities in patient population and omissions of crucial data such as patient body temperature or time of submersion until effective CPR started. Recent studies suggest that complete flaccidity including lack of sponotaneous ventilation at ICV admission (flaccidity at ER admission may change after CPR which improves prognosis) is associated with by far the worst prognosis. In Allman's series of warm-water near-drowning, of 37 patients in flaccid coma at ICU admission, 26 died and 11 survived in a persistent vegetative state despite CRT in most. The use of CRT in such patients may not be appropriate.

From an ICU management standpoint, we are left with a decision regarding CRT in comatose neardrowning victims who demonstrate any motor activity to painful stimulus. Increasing experience suggests that elevated intracranial pressure even when controlled is associated with extremely poor prognosis.17,18,29,31 The duration of complete cerebral ischemia with lack of oxygen and glucose delivery determines the extent of irreversible cellular damage. Cytotoxic cerebral edema and elevated intracranial pressure is a manifestation of irreversible cellular damage and would be expected to be associated with a poor outcome even if controlled. Presently, the pendulum swings away from CRT in the management of comatose (decorticate, decerebrate, flaccid) near-drowning victims. Only a prospective, randomized, multi-institutional study will resolve this important controversy.

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Look-Alike and Sound-Alike **Drug Names**

BENJAMIN TEPLITSKY, R. PH. Brooklyn, N.Y.

Look-alike and sound-alike drug names can be misinterpreted by a nurse reading doctors' orders or by a pharmaeist compounding physicians' prescriptions. Such misunderstandings can result in the administration of a drug not intended by the prescriber. Awareness of such look-alike and sound-alike drug names can reduce potential errors.

Category: Brand Name:

Generic Name: Dosage Forms:

Category: Brand Name:

Generic Name: Dosage Forms: ANTRENYL

Gastrointestinal

Oxyphenonium Bromide Anthralin Tablets

AMITRIPTYLINE Antidepressant Amitril, Parke-Davis

Elavil, MSD Endep, Roche Amitriptyline HCl Tablets, Injection

ANTHRALIN

Antipsoriatic Antrenyl Bromide, Ciba Anthra-Derm, Dermik

Lasan, Stiefel

Ointment, Cream

AMINOPHYLLINE Bronchodilator Somophyllin, Fisons

Aminophylline

Tablets, Elixir, Suppositories, Injection, Rectal solution, Oral

solution

Principles of Mechanical Ventilation

Critical Care Medicine

BRIAN M. GROSS, M.D.¹ DAVID J. POWNER, M.D.² Indianapolis

■HE USE OF mechanical devices for ventilatory support has interested researchers ever since Vesalius used a bellows to inflate the lungs of animals in 1543. This paper will outline general principles for initiating mechanical ventilation and potential hazards to the patient inherent in the selection of the various ventilator parameters. The physician ordering respiratory care via a mechanical ventilator must be conversant with the effects of those several variables ordered and also of the settings usually not ordered but adjusted by the respiratory therapist or nurse.

Before discussing specific ventilator settings, some consideration should be given to physiologic alterations in the ventilated patient. Ventilators generate a positive pressure to force gas into the lungs (as compared to normal inspiration where air moves in because the patient generates negative airway pressure). This has three potential dangers. The increased airway pressure could rise to such a level as to cause lung rupture. Secondly, the elevated airway pressure also increases intrathoracic

pressure, causing resistance to venous return and other hemodynamic changes which may potentially lead to hypotension. Thus, positive pressure ventilation can have direct effects on cardiac output. The third area of potential harm involves changes in the inspiratory and expiratory times (I:E ratio), which can cause inadequate gas exchange, air trapping and changes in intrathoracic pressure.

Types of Ventilators

Ventilators commonly used today are divided into three basic types. Time-cycled devices deliver the tidal volume during a preset inspiratory time period. The airway pressure generated will be a function of the tidal volume, inspiratory time chosen, and pulmonary compliance and resistance. Pressure-cycled devices deliver gas until a preset airway pressure is achieved. Tidal volume varies with changes in lung compliance or airway resistance with these ventilators. A volume-cycled ventilator delivers a predetermined tidal volume with each breath. The positive pressure required and hence peak airway pressure, intrathoracic pressure, and I:E ratio can vary from breath to breath depending on the several variables discussed below. Most ventilators used for mechanical ventilation are volume-cycled, and therefore the following discussion is limited to such devices.

Ventilator Settings

Oxygen: Inspired O. can be delivered in concentrations from 21% (ambient air, FI. 0.21) to 100% (FI. 1.0).

Patients requiring emergent ventilator support are often begun on 100% O₂, but the dangers of O₂ toxicity mandate that the O₂ concentration be decreased to the lowest level (especially less than 50%) consistent with adequate oxygenation (PaO₂ over 55; arterial O₂ saturation over 90%) as quickly as possible.

Tidal Volume (V_{τ}): Although a normal V_{τ} during spontaneous breathing is about 500 cc, prevention of alveolar collapse and atelectasis in the ventilator patient has led to a high-volume, low-frequency technique. Tidal volumes are usually set at 10-15 ml/kg (tean body weight). The tidal volume selected directly influences peak airway and intrathoracic pressures generated.

Sigh/Sigh Volume: Although technically still available, sighs are generally not used, having been made obsolete by the high V_+ low-frequency technique. If used, sigh volumes (2-3 times V_{\pm} at a rate of 1-15/hr.) will substantially affect airway and intrathoracic pressure.

Frequency, I:E ratio: Initial respiratory frequencies (f) are usually set at 8-10 breaths/min. when a high V₁ is used. This rate determines the respiratory cycle time, or the time in which both inhalation and exhalation occur. For example, a frequency of 10 breaths/min means a 6 second cycle time. The inspiratory:expiratory (I:E) ratio expresses how the cycle time is divided between inspiration and exhalation. Usually, this ratio is 1:2 to 1:3, and ventilators often alarm if the ratio reaches 1:1.

The I:E ratio is important because exhalation is usually and most desir-

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TABLE 1 Possible Causes Increased Airway Pressure

- Kinked ET tube
- 2. Occlusion of airway by secretions
- 3. Pneumothorax/tension pneumothorax
- 4. Mainstem intubation/migration of ET Tube
- Decreased lung compliance (progressive fibrosis, incr. lung water)

ably a passive process. If the expiratory time is too short and exhalation is incomplete, the lungs may remain partially inflated when the next machine breath is inhaled, leading to over-inflation and alveolar distention. Alternatively, the patient may be forced to use accessory muscles to speed exhalation, thus incurring additional work of breathing. A shortened expiratory time may be particularly detrimental in patients with asthma or chronic obstructive lung disease, as such patients have an inherent impedance to exhalation. The I:E ratio is also important since a prolonged inspiratory phase raises the mean airway pressure.

Airway pressure/Flow rates: The positive pressure required to inflate the patient's lungs is a function of the volume of gas delivered (V_r), the time in which it is delivered (inspiratory time), airway resistance, and the compliance of the patient's lungs and thorax. The flow rate at which gas is delivered depends on the V_r and inspiratory time.

Different ventilators offer various ways to control these variables. On the Bennett MA-1, the operator selects (or the physician orders) the rate, $V_{\rm T}$, and peak flow rate (usually 40-60 L/min.), which indirectly determines I:E ratio. Airway pressure is thus a dependent variable and can be viewed on the control panel. The Siemens Servo ventilator lacks a flow rate dial; instead, one selects f, minute volume

(hence $V_{\rm F}$), and the percentage of in spiratory time, (from 25-50% of the cycle time). Thus, the I:E ratio is preset and flow rate and airway pressure are passively determined.

Any combination that demands a short inspiratory time will, for any given $V_{\rm t}$, increase flow rate. As flow rate is directly or indirectly increased, airway and intrathoracic pressure rise, possibly subjecting the patient to barotrauma or hemodynamic change.

Pressure Alarm: The pressure alarm is used to detect high airway pressures. It is usually set about 10 cm H₂O above the patient's current peak airway pressure. Sudden increases in airway pressure can signal a variety of problems (Table 1) that may be life-threatening. If the pressure limit is reached, the ventilator cycles to the expiratory phase and the rest of that machine breath never reaches the patient. The patient who is constantly "pressure limiting" requires prompt evaluation.

Mode of Ventilation (Figure 1): The ventilation mode determines how the patient and ventilator interact:

(1) Controlled ventilation (CV). The machine delivers a preset number of breaths at a preset $V_{\rm T}$. No additional gas is available to the patient beyond that delivered by the machine. The patient is in a closed circuit between machine breaths, and spontaneous respiratory efforts do not open a system to bring additional fresh gas to the patient. Thus, there is no compensatory mechanism in place if ventilation needs change.

(2) Assist-Control (AC). A back-up rate is selected so that, in case of apnea, a minimal number of machine breaths will be delivered. However, the ventilator also senses the patient's inspiratory efforts (with the operator able to control the ventilator's sensitivity) and provides a machine V_{\perp} each time the patient initiates a breath. This allows the patient to set his/her own respiratory rate to match changing requirements.

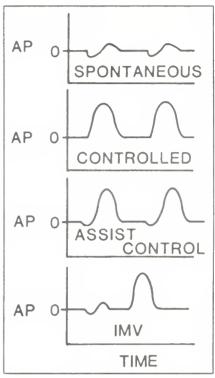


FIGURE 1: Ventilations Modes: Changes in Airway Pressure (AP) vs. time in spontaneous ventilation, controlled ventilation, AC (note patient initiated machine $V_{\rm I}$), and IMV (note both patient and machine breaths in parallel. O = Atmospheric Pressure

On the other hand, patients with altered respiratory drive (fever, anxiety) may exceed their real requirements and reach a dangerous respiratory alkalemia.

(3) Intermittent Mandatory Ventilation (IMV). Originally introduced as a new weaning method, IMV is now often used as the primary mode of ventilation. The ventilator delivers a preset number of machine breaths, between which the patient can breathe spontaneously from a reservoir gas circuit at his/her own tidal volume. Synchronized IMV (SIMV) is a circuitry modification in which the ventilator will synchronize its tidal volume delivery with the patient's inspiratory effort (within a certain temporal window). The idea is to pre-

TABLE 2 Initial Ventilator Settings

Tidal Volume 10-15 ml/kg Resp Rate 8-12 O. Conc. 100% (to start) Flow rate 40-60 L/min

Airway pressure alarm; 10 cm above peak airway pressure

Mode usually IMV or AC

vent a machine breath from being forced into the airways as the patient completes a spontaneous inspiration. How much of an improvement this really represents is debatable. In the apneic patient, equal rates of CV, AC, or IMV all yield the same result, and any mode can be used in this situation.

Other options available include:

PEEP: Associated with increases in FRC, Positive End-Expiratory Pressure has an important place in management of ARDS and other states of acute lung injury. PEEP often allows adequate blood oxygenation with lower oxygen concentrations. Some authors advocate the use of 5 cm PEEP in almost all patients, especially those with resolving lung injury (physiologic PEEP). Potential disadvantages include increased intrathoracic pressure and, as a function of lung compliance, increased airway pressure. PEEP's impedance to exhalation may or may not exacerbate air trapping in cases of obstructive lung diseases. PEEP is usually ordered in the range of 5 to 20 cm H.O; super-PEEP (over 20 cm H.O) may sometimes be needed. PEEP is usually adjusted in increments of 2-3 cm H₂O, and patients can be extubated from a level of 5 cm H₂O.

Expiratory retard: This modification introduces a resistance to expiratory flow analogous to pursed lip breathing in emphysema. The difference between this and PEEP is illustrated in Figure 2.

Inspiratory Hold (Pause/Plateau): This option stops the cycle at endinhalation, pausing for a period of time measured in seconds or as a percentage of inspiratory time (Figure 3). This technique increases mean airway pressure without changing peak airway pressure. Expiratory retard and inspiratory hold do not need to be used in most patients, but have been advocated to treat hypoxemia.

Attention to the factors outlined in $Table \supseteq$ should suffice for initial ventilator settings. The efficacy of the settings can only be assessed by determining if the ventilator satisfies the patient's ventilatory needs. Arterial blood gases (ABG) will document adequate oxygenation and CO. elimination. ABGs are usually obtained about 30 minutes after a change in ventilator settings. Newer technologies such as ear oximetry, intraarterial oximetry, or PA catheters with continuous oximetry lessen dependence on ABGs, although none of these assesses CO. elimination or arterial pH.28 In addition to blood gases, attention must also be directed to the patient's work of breathing and appropriate adjustments made to minimize patient exertion.

Importantly, therefore, mechanical ventilators, when properly adjusted, can provide comfortable, as well as life-saving, respiratory support. These goals can be provided without unto-

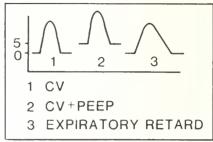


FIGURE 2: PEEP raises the baseline system pressure; expiratory retard delays the return to atmospheric pressure.

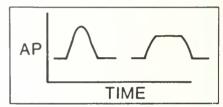


FIGURE 3: Illustration of difference between a normal ventilator breath and inspiratory hold.

ward complications by a knowledgeable physician working closely with nurses and respiratory therapists.

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Giardiasis: A Community Problem

PEG RAMEY, R.N. STAN REEDY, M.D. Elkhart

Elkhart County
Health Department
Conducts Study
to Determine
Incidence of
Giardiasis; Finds
That Many Cases
Are Transmitted
Person-to-Person

IARDIA LAMBLIA is the most commonly recovered parasite In stool samples submitted in the United States. Publicized waterborne outbreaks of giardiasis in California, Colorado, Washington and Pennsylvania have been linked to the pollution of mountain stream water or stored municipal water supplies by beaver colonies. Because the literature frequently refers to these outbreaks, physicians in non-mountain areas or beaver-free communities may overlook giardiasis as a diagnostic possibility. Community patterns for giardiasis need to be studied and the role of person-to-person transmission better understood. A study by the Elkhart County Health Department for these purposes is described in this report.

The Organism

Giardia lamblia is a protozoan organism that exists in both the trophozoite and eyst forms. The eyst is capable of surviving three months or more in cold water at 8° C. Survival is shortened to one month in water at 21° C and to four days at 37° C.2 Cyst formation does not take place outside the host. The trophozoal stage is not considered infective. Mammalian hosts include beavers, raccoons, bighorn sheep, mule deer, dogs, cats and humans. Laboratory animals such as rats, guinea pigs and gerbils have also been experimentally infected with human source Giardia lamblia.3

Clinical Manifestations

Most persons seeking medical attention for illnesses finally diagnosed as giardiasis report diarrhea of two to four weeks duration with loose, foamy stools, abdominal cramping, nausea, anorexia and occasionally, fever. Fifteen to thirty pound weight

losses have occurred, along with signs of nutritional deficiency. Milder eases may report only a constant "bloated" feeling or persistent abdominal pain with no diarrhea. Some children have gone through extensive evaluation for appendicitis before giardiasis was finally diagnosed. An asymptomatic "carrier" state does exist; these people may only be diagnosed when tests are run on household contacts of persons with symptomatic giardiasis.

Transmission

Transmission of giardiasis depends on the ingestion of eysts that are excreted in the feces of a human or animal host. As few as 10 cysts can infect humans. The incubation period ranges from one to four weeks. Persons with acute diarrhea due to giardiasis are excreting the trophozoite stage, so are less likely to be infective. Untreated, recovering giardiasis victims or asymptomatic carriers are much more likely to be infective because slower intestinal transit permits the formation of cysts. The transmission risk increases when persons practice poor hygiene habits, or if assistance in toileting is required. The risk of acquiring giardiasis also increases for persons who drink untreated surface water. Male homosexuals have been found to be at increased risk.

One Health Department's Experience

From 1980 through 1983 more than 100 persons were reported to the Elkhart County Health Department with stools positive for Giardia lamblia. This is more than the combined totals of Salmonella and Shigella infections for that time period. *Table 1* lists enteric diseases reported for those four years.

Giardiasis is not on the official re-

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portable disease list for Indiana, although reports are requested. Because not all counties are reporting giardiasis, state figures do not reflect the same comparison pattern for these three diseases. Reports of giardiasis to the State Board of Health have increased annually, with about 400 cases being reported in 1983, compared to about 200 in 1982. Actual community incidence is unknown.

In Elkhart County, reports of giardiasis were individually investigated by the Communicable Disease Section Nurses, following permission from the physician involved. Background interviews were obtained from most families. Only a few of those infected were Indochinese refugees. Two persons admitted drinking Colorado stream water while backpacking, one admitted drinking Canadian lake water while on a fishing trip. Five families had camped in Texas, Florida or Missouri and one girl accidentally swallowed water during water slide use in Iowa. All these cases were judged to be imported to Elkhart County.

The majority of persons with giardiasis in Elkhart County had no travel history outside Elkhart or surrounding counties. An attempt was made to document a common source for this group. No such source has been identified; cases were reported from both rural and municipal areas scattered throughout the county. No cluster of cases implicated a specific water supply. Both public and private drinking water in this area is supplied by deep

TABLE 1					
Enteric Disease Repor	ts to Elkhart Co	unty Hea	alth Depa	irtment,	1980-1983
	1980	1981	1982	1983	Total
Giardiasis	12	26	33	35	106
Salmonella	15	12	22	23	72
Shigella	3	4	2	6	15

wells. Sand filtration effectively screens out Giardia lamblia cysts, so well water has not been considered a source. No outbreak was associated with an organized day care center, although some babysitting situations did prove to be the link between cases.

Two items did emerge from interviews as potential risk factors:

- 1. Use of recreational water by a family member.
- 2. Possible person-to-person transmission, especially in homes with children less than age 3.

Recreational water use was named by a number of families and included swimming, boating or water skiing in area lakes and rivers. Fishing or camping near a lake was often reported. Water use sites included bodies of water in Elkhart County as well as those within a 50-mile radius of Elkhart County, Lakes, ponds, the St. Joseph and Elkhart Rivers, and public swimming pools, both indoor and outdoor, were named. Since no practical laboratory method is available to test such waters for Giardia lamblia eysts, this risk factor remains speculative and in need of further investigation.

During 1982 and 1983 attention was

directed to the potential for transmission within a family. The Communicable Disease Section Nurses urged that all family members be tested if one member was found to be positive for giardiasis. Results of this focused study follow:

Method-Reports of Giardia lamblia positive stool examinations were solicited from the laboratories serving the two local hospitals. Permission to interview each symptomatic case was obtained from the physician involved. Families were interviewed, given information sheets on giardiasis and offered Indiana State Board of Health 4-A mailing containers for ova and parasite specimens. If the family agreed to testing, one container per person was then issued for limited screening purposes. (A more thorough diagnostic work-up might include three or more stool samples, or other diagnostic tests.) Containers were offered for post-treatment check, because current treatment regimens are judged to be only 80% to 90% effective.2 (For a few families, all containers were for treatment checks, since some physicians preferred to treat the whole family if one member was positive.) Samples were mailed

TABLE 2						
Results of 18 Household Groups Tested for Giardiasis Following Infection						
Diagnosed in One Member						

Age range of symptomatic index case	Number of Cases	House- holds tested	Households with addition- al positives	Total additional positives	Age range of additional total positives
Child 0-3	10	8	5	10	10
Child 6-10	1	1	1	1	9
Child 11-16	5	3	0	0	1
Adult 17-64	21	6	6	14	5
	$\overline{37}$	18	12	25	$\overline{25}$

to the Indiana State Board of Health Laboratory by the patient, parent or babysitter. Results were returned by mail within 10 days to the Elkhart County Health Department. Positive results were phoned and mailed to physicians as they were received.

Results - During this two-year period, 37 households were contacted by the Communicable Disease Nurses. Twenty-five of these households contained children less than 3 years of age. Containers were issued to 26 households or contact groups (babysitters or grandparents). Eighteen groups eventually submitted samples. Specimens from 113 persons were sent to the Indiana State Board of Health laboratory. Twenty-five additional persons from 12 household groups were found to be positive for Giardia lamblia. Table 2 summarizes the numbers and age ranges of additional stool-positive persons in households with a symptomatic index case.

Discussion

This limited study suggests that transmission of giardiasis within a family occurs, especially if young children are in the home. Symptoms are not a reliable indicator of giardiasis as many of the household contacts positive for Giardia lamblia were not

having symptoms. Information published by the American Public Health Association and the Indiana State Board of Health indicates that asymptomatic household earriers are a source of giardiasis transmission within a family. Treatment for such individuals is important.

Recreational water use is still considered a potential risk factor. Giardiasis may be introduced into a family by the fact that young children are more likely to swallow lake or pool water containing Giardia lamblia cysts and then transmit the infection later to care givers who handle soiled diapers or help with toileting. One reeent study linked a swimming class attended by diapered children to an outbreak of giardiasis in the community.6 It should be noted that usual chlorination techniques do not eliminate Giardia lamblia cysts in either drinking or pool water. Some day care/ babysitting situations are also a risk factor as 2- and 3-year-olds typically do not wash hands after bathroom use unless closely supervised.

The Elkhart County Health Department plans to continue interviewing diagnosed giardiasis cases and urging family tests. So far in 1984, similar results in families and babysitters have been obtained. Information on giardiasis has been mailed to

all area physicians at least twice during this study, possibly prompting more consideration of this diagnosis.

Attention nation-wide has been focused on the growing incidence of enteric disease, including giardiasis associated with day care centers. Large scale studies underway in other states may result in specific recommendations regarding giardiasis in day care settings.⁷

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Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricu-lar conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses

Interval can be seen at higher doses In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect, cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been aftected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by dittazem.

or slightly reduced by diltiazem Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20% In a study involving single oral doses of 30D mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block Dilitazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, dilitazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not

usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltazem is absorbed.

from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40% CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 7D% to 8D% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 3D to 12D mg of CARDIZEM result in detectable plasma levels within 3D to 6D minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 35 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 50 mg are given, a 12D-mg dose gave blood levels three times that of the 6D-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem

INDICATIONS AND USAGE

Angina Pectoris Due to Coronary Artery Spasm. CARDIZEM

is indicated in the treatment of angina pectoris due to coronary artery spasm CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).

Chronic Stable Angina (Classic Effort-Associated Angina).

CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance. There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this.

combination in patients with impaired ventricular function or conduc tion abnormalities

CONTRAINOICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 9D mm Hg systolic)

1 Cardiac Conduction. CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recover. time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for D 48%). Concomitant use of AV DIOCK (SIX OT 1243 PAIRENTS FOR 19 48%). Concomitant use of dilitiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of dilitiazem.

Congestive Heart Failure. Although dilitiazem has a negative

inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should

be exercised when using the drug in such patients **Hypotension.** Decreases in blood pressure associated with

CARDIZEM therapy may occasionally result in symptomatic

Acute Hepatic Injury. In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes,

however, these changes were reversible with continued dosing **Drug Interaction.** Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM (See

WARNINGS)

Controlled and uncontrolled domestic studies suggest that con-comitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxir levels up to 2D%

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed

Pregnancy. Category C Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 2D times the human dose or greater.

There are no well-controlled studies in pregnant women, therefore, se CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation

Pediatric Use. Satety and effectiveness in children have not been established

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than

that reported during placebo therapy

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition in many cases, the relationshm to CARDIZEM has not been established The most common occurrences, as well as their frequency of presentation, are edema (2.4%). headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%) asthenia (1.2%), AV block (1.1%), in addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence

Flushing, arrhythmia, hypotension, bradycar Cardiovascular dia, palpitations, congestive heart failure

syncope

Paresthesia, nervousness, somnolence tremor, insomnia, hallucinations, and amnesia Constipation, dyspepsia, diarrhea, vomiting mild elevations of alkaline phosphatase, SGOT

SGPT and LDH Dermatologic Other

Nervous System Gastrointestinal

Bradycardia

High-Degree AV Block

Pruritus, petechiae, urticaria, photosensitivity Polyuria, nocturia

The following additional experiences have been noted:
A patient with Prinzmetal's angina experiencing episodes o

A patient with Prinzmetal's angina experiencing episodes or vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mi dose of CARDIZEM

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM erythema multiforme, leukopenia, and extreme elevations of alkaline phosphatase, SGO SGPT, LDH, and CPK. However, a definitive cause and effect betwee these events and CARDIZEM therapy is yet to be established

OVEROOSAGE OR EXAGGERATEO RESPONSE

Overdosage experience with oral diltiazem has been limiter Single oral doses of 30D mg of CARDIZEM have been well tolerate by healthy volunteers. In the event of overdosage or exaggerate response, appropriate supportive measures should be employed addition to gastric lavage. The following measures may be considered:

Administer atropine (0.60 to 1.D mg). If ther

is no response to vagal blockade, administe isoproterenol cautiously Treat as for bradycardia above Fixed high degree AV block should be treated with ca

diac pacing Administer inotropic agents (isoprotereno Cardiac Failure dopamine, or dobutamine) and diuretics. Vasopressors (eg., dopamine or levarterent Hypotension

bitartrate).

Actual treatment and dosage should depend on the severity of th clinical situation and the judgment and experience of the treating

The oral/LD_{so}'s in mice and rats range from 415 to 74D mg/k and from 560 to 81D mg/kg, respectively The intravenous LD_{so}'s these species were 60 and 38 mg/kg, respectively The oral LD_{so}' dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not know but blood levels in excess of 800 ng/ml have not been associate with toxicity

OOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atheroscierotic Con nary Artery Disease or Angina Pectoris at Rest Due to Cornary Artery Spasm. Dosage must be adjusted to each patient needs. Starting with 30 mg four times daily, before meals and shedtime, dosage should be increased gradually (given in divide doses three or four times daily) at one- to two-day intervals unloptimum response is obtained. Although individual patients may be a supported to the control of the cont respond to any dosage level, the average optimum dosage rang appears to be 180 to 240 mg/day There are no available data concer ing dosage requirements in patients with impaired renal or hepat function if the drug must be used in such patients, titration should t

carned out with particular caution.

Concomitant Use With Other Antianginal Agents:

 Sublingual NTG may be taken as required to abort acu-anginal attacks during CARDIZEM therapy
 Prophylactic Altrate Therapy — CARDIZEM may be safe coadiministered with short- and long-acting nitrates, but the have been no controlled studies to evaluate the antiangin effectiveness of this combination.

3 Beta-blockers. (See WARNINGS and PRECAUTIONS)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NE DB88-1771-47) and in Unit Dose Identification Paks of 100 (NE 0B88-1771-49) Each green tablet is engraved with MARION on or side and 1771 engraved on the other CARDIZEM 6D-mg scor tablets are supplied in bottles of 100 (NDC DB88-1772-47) and in United the proceedings of the process of the pro Dose Identification Paks of 1DD (NDC DD88-1772-49). Each yell tablet is engraved with MARION on one side and 1772 on the other states of the state o Issued 4/1/I

Another patient benefit product from



Smoking Withdrawal

An Evaluation of Its Role in the Total Effort to Stop Smoking

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Abstract

This report presents the case that smoking withdrawal clinics and similar efforts to assist the habituated smoker are cost-ineffective, for the following reasons. Even the modest 25 to 30% remission rate of the average withdrawal effort is probably inflated by misleading self-reports. Reported abstinence over a given time period may not be continuous. U.S. Department of Agriculture data do not support the contention that "30 million Americans have given up smoking." Regardless of the number of quitters, the Surgeon General has estimated that 95% have quit on their own, without formal assistance. Few smokers elect to participate in formal withdrawal efforts. It would be more cost-effective to place a greater emphasis on preventive efforts or media efforts in which large numbers of smokers can be reached with a minimum of cost.

RADITIONAL PUBLIC HEALTH policy is based on an obvious logic: prevent if we can but treat if we must. In the case of the human malignancy known as cigarette smoking, the primary treatment source is the so-called withdrawal or "quit" clinic. Such clinics have been sponsored continuously for the past two decades by voluntary health organizations, church groups, universities, hospitals, medical clinics, by commercial enterprises and by researching scientists and clinicians. A variety of techniques and combinations of techniques have been employed.

Ideally, the effort to find a prophylaxis and the effort to improve existing treatment tactics until such prophylaxis becomes available should not be viewed as competing endeavors. But in the sense that resources are never unlimited, treatment and prevention efforts must of necessity compete to some extent. The significant question concerns the proportion of resources that should be allocated to each effort.

Though prevention is primary in public health policy, a highly effective treatment program, as shown by a cost-benefit analysis, would surely be worth a fair share of the available resources. On the other hand, an ineffective treatment procedure, one that yielded a relatively small number of cures in relation to the output of treatment effort, a low benefit per cost, is cost-ineffective and should not command much of the available resources. In such an instance, the lion's share, perhaps a share for a whole pride of lions, should be allocated to prevention.

Unfortunately, recourse to a traditional cost-effectiveness/cost-benefit analysis¹ of quit clinics is not possible. As Perloff *et al.*² point out, "It is no trivial task to identify the output of a complex institution . . . it is even more complicated to place a monetary value on thse outputs" (page 579). It is even more difficult, I might add, when accurate data are missing or otherwise unavailable.

Cost-effectiveness data have been reported for a smoking control program developed by Kaiser-Permanente.³ Allegedly, a 62% abstinence rate was obtained at a two-year follow-up at a cost per patient of \$22.48. Unfortunately, this is merely a statement in the report; the fiscal mathematics are not given. Furthermore, the group was quite small. Only 49 subjects completed the program. Finally, the evaluation was hardly independent; its author was at that time chief psychologist for Kaiser-Permanente's program.

A cost-effectiveness survey of smoking withdrawal programs by Green *et al.*⁴ is on the right track but was badly hampered by missing data. Less than half of the programs surveyed contained enough data on methods and results to provide a cost-effectiveness estimate. Even among those studies, much follow-up data are lacking.

The average cost-effectiveness at six-month follow-up was \$64 per person for programs using aversive conditioning methods, \$430 for hypnosis and \$3 for behavior modification programs other than aversive conditioning. (As Green *et al.*⁴ note, this last deflated figure is a function of using

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graduate students instead of professional persons.) The estimates appear to be based entirely on man hours. Green *et al.*; point out that other important costs in a large scale program such as recruitment, overhead, training of clinic leaders, materials development, follow-up and overall evaluation, were not calculated.

Despite a noble effort, the only firm conclusion that can be drawn from the Green *et al.* work is that group methods are likely to be more cost-effective than one-on-one methods.

Let's approach the assessment of treatment from another angle. A number of summaries of the outcome of formal smoking withdrawal efforts have been published over the past 15 years. 14 The general conclusion is that outcome results are typically disappointing. Without exception, data summaries report the "extinction curve" first noted by Hunt & Matarazzo,15 a phenomenon that smoking cessation shares with withdrawal from alcohol and drugs of addiction.16 At the conclusion of the smoking withdrawal program, a substantial proportion of participants will report that they have given up smoking completely. Thereafter, however, a follow-up will invariably indicate that most of the quitters have come recidivists. As Evans et al.13 point out, "Study after study suggests that it is clearly possible to persuade smokers to stop for varying lengths of time during a period of several months. The same studies (where adequate evaluative data are presented) either directly or indirectly support the notion that most former smokers will not continue to abstain from smoking" (page 238). Typically, about two-thirds of the temporary abstainers will have backslid by the end of three months. At the end of the year, about 30% of the original participants still report abstinence. There is, of course, some variation around this mean estimate but certainly no more than would be attributed to sampling error. Long range follow-up studies¹⁷ suggest that the proportion of abstainers may shrink to less than 20% in five years.

Valid cost-effectiveness data must be based upon valid statements of cost and valid statements of outcome. There is excellent reason to believe that even the modest 25 to 30% success rate of most smoking withdrawal clinics is inflated. Usually, outcome data are self-reports by clinic participants. Over the past seven years, there have been a considerable num-

Even the Modest 25-30% Success Rate of Most Smoking Withdrawal Clinics Is Inflated ...

ber of withdrawal efforts in which self-reports were compared with various objective physiological indicators of smoking behavior.1829 Without exception, these reports indicate that the number of persons reporting that they were abstaining from smoking is greater than the number who were found to be abstinent by physiological measurement. The discrepancies vary from 10% to as much as 40%. Thus, it would appear that 5 or 6% of withdrawal clinic participants who report abstinence are either deliberately dissembling or, as Vogt³⁰ has recently suggested, are deceiving themselves.

Two further methodological considerations argue that outcome data are likely to be overly optimistic. The findings of Burgess and Tierney³¹ strongly suggest that in a follow-up by mail, a considerably higher percentage of nonrespondents are likely to be smokers. Thus, if nonrespondents were to be included in the outcome data, the success rate would be lower.

It is usually assumed that a withdrawal clinic participant who is found to be abstinent at any point in time has been completely abstinent up to that point. Evans and Lane³² report a rare study in which this assumption was tested. They found that 25% of the withdrawal clinic participants were abstinent at the end of a year. However, a full third of that 25% had not been *continuously* abstinent from clinic termination to follow-up. The exact significance of this inconsistency for outcome data is not clear but it is plain that the effect must be negative.

Perhaps the most damaging blow to withdrawal clinics was struck a few years ago by Warner.33 He analyzed national cigarette consumption in the United States extrapolated from surveys of smoking behavior and from actual cigarette consumption based on production and sales data provided by the U.S. Department of Agriculture. He found that between 1964 and 1975, cigarette consumption should have increased by only 4.74%, according to the surveys of smoking behavior, a very heartening finding considering that the smoking-age population increased by almost 20% during that same period. However, USDA data indicated that smoking consumption increased by 18.46% in that same period. This is nearly four times as great an increase as should have been found on the basis of selfreport data. Sadly, one is forced to conclude that if hard facts are used as a basis, cigarette smoking in the United States has not decreased very much since 1964.

Nevertheless, one reads repeatedly that 30 million Americans have given up smoking since the epochal report of the Surgeon General of the United States in 1964. The origin or basis of this estimate is obscure and it is surely questionable. Even if it were completely correct, the argument against the quit clinic is unaffected. The important estimate by the Surgeon General is that "Ninety-five per cent of those who have quit smoking have done so without the aid of an

organized smoking cessation program ..." The National Cancer Institute reached the same conclusion some years ago. 35

So 5% of 30 million = 1,500,000 exsmokers are the nearly 20-year yield of the withdrawal clinics—about 80,000 or 85,000 persons per year. How many withdrawal clinics are required to reach this product?

A published estimate of the number of quit clinics annually in the United States is not available. The American Cancer Society reported in 1975 that its divisions sponsored 2,176 quit clinics, reaching 32,638 smokers. The success rate is noted as 25 to 30%. Assuming the complete accuracy of these figures, each clinic produced four or five new ex-smokers. There must have been between 17,500 and 19,000 quit clinics to yield 80,000 ex-smokers per year—relatively speaking, a mountain of labor for a mouse of product.

Participants in quit clinics are volunteers. Several studies show that even when participation in the clinic is facilitated, only a small percentage of smokers will take advantage of the opportunity.36-37 Lehrer38 reported from Israel a few years ago that 42,000 smokers wrote to the Ministry of Health for a booklet on smoking withdrawal but only 400 of them attended a clinic. As Kanzler et al.36 remark, such self-selection is seriously damaging to the withdrawal effort. They go on to note the simple fact that a 25% withdrawal rate would be worthy if two-thirds of smokers participated, but even a very high success rate will have little impact on the smoking problem if few smokers are reached. That seems to sum up the withdrawal clinic in a few words.

One further consideration: Lehrer suggests that quit clinic participants tend to regard themselves as addicted and thus ill in the medical sense in contrast to habituated. They seek a medical model treatment in which the full responsibility for withdrawal is shifted to the clinical personnel.

Thus, Lehrer concludes, withdrawal clinic participants have the poorest prognosis for quitting among smokers. In support, Schachter has pointed out that the professional consensus that disorders like obesity, drug addiction and smoking are particularly resistant to change is based on self-selected patient groups. Within groups not seeking professional help, many permanent self-cures occur (i.e., 95% of those who have quit smoking).

The conclusion should be obvious.

Smoking Withdrawal Clinics Are Seriously Cost-ineffective and Should Be Abandoned . . .

Smoking withdrawal clinics are seriously cost-ineffective and should be abandoned as a tactic in the war on cigarette smoking. Of course, research efforts should continue but there is a definite difference between developing new programs and simply repeating the old ones.

For example, the recent report by the Ontario Council of Health Task Force on Smoking 11 calls for a withdrawal effort for smokers as a part of "a need for balance" in a provincial smoking control program. But the research aspect of this prong of the attack is clearly salient. The specific recommendation calls for developing "a limited number of demonstration projects" (p. 18). The literature review on which this recommendation is based notes that "continuing research is necessary" and "further development and evaluation are needed" (p. 104). The emphasis on research is

The idea that the smoking withdrawal clinic has at best an insignificant place in the armamentarium of the anti-smoking forces is not new. It was first stated a decade ago by the Advisory Committee on Smoking & Health to the Swedish National Board of Health and Welfare, in its proposal for a national smoking control program:

"... smoking withdrawal clinics can provide only a small fraction of the necessary change and therefore cannot be the relevant instrument to achieve the wide scale decrease in the percentage of smokers in the total population."⁴²

And more recently by Evans and his associates:

"After examining the many current trends in smoking research, it is increasingly evident that focusing primarily on programs to persuade already addicted smokers to stop smoking will be of only limited value... The number of individuals who can be expected to stop smoking permanently... may be 'bottoming' out... in other words... a certain percentage of 'hard-core' addicted smokers are literally unable to stop smoking permanently, regardless of the control program to which they are exposed."

Two efforts in Finland suggest that the smoking withdrawal effort might benefit by a union with public television. 13 A televised withdrawal clinic that would have an outcome success rate of .5% as in the Finnish study would be enormously cost-effective. But television can also be used for prevention as it was in the United States for several years. The paramount conclusion is that we need to turn many, many more of our dollars and our people hours to prevention, essentially, the education of our youth. We need to label school rooms and curricula, not cigarette packages. And let's begin by admitting that the conventional smoking withdrawal clinic is being used largely to advertise its sponsor, not to impact the smoking control movement.

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ALCOHOLISM

A Diagnostic and Therapeutic Challenge

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In my judgment such of us who have never fallen victims (to alcoholism) have been spared more by the absence of appetite than from any mental or moral superiority over those who have. Indeed, I believe if we take habitual drunkards as a class, their heads and hearts will bear an advantageous comparison with those of any other class.

-Abraham Lincoln

LCOHOLISM is a complex disorder which has plagued societies since the advent of history. References to the intoxicating and destructive potential of alcohol have been found in ancient Egyptian hieroglyphics and in the writings of the Mesopotamians who proposed a remedy for the common "hangover." Today, we also can attest to the devastating consequences of alcoholism; the problem of alcohol abuse presents a continuing major challenge to medical research, to the practicing clinician, and to our system of public health.

In 1984 only coronary artery disease and cancer claimed more lives than alcoholism and its complications.1 During the last quarter century we have witnessed impressive advances in the understanding and treatment of coronary disease and neoplasia. The results have been both a significant reduction in morbidity and mortality from heart disease and increasing numbers of remissions and even cures from cancer. Unfortunately, we have not observed a similar impact on alcoholism, a disorder which presently affects an estimated 14 million Americans. Cirrhosis has now surpassed diabetes as the fifth leading cause of mortality.2 Twenty to twenty-five per cent of patients in our hospitals have medical problems attributable to alcohol. A quarter of a million Americans have died during the last decade in motor vehicle accidents involving alcohol, and suicide is 30 to 50 times more common in the alcoholic population. The resulting annual economic loss, including those due to utilization of the judicial system, accidents, job absenteeism, lost productivity, as well as medical costs is approaching \$50 billion.³ The disruption of family integrity which frequently leads to divorce, unemployment, and child abuse and delinquency is perhaps even more significant, but less easily measured. The magnitude of human suffering due to alcoholism is truly staggering.

This paper suggests some ways of approaching alcoholism, and provides some practical suggestions to assist the clinician in identifying and helping alcoholic patients. Specifically we hope to accomplish this by:

- 1. Providing a workable definition of alcoholism.
- 2. Emphasizing the importance of the "disease concept" and summarizing the evidence for its acceptance and usefulness.
- 3. Encouraging physicians and health care professionals to examine their own attitudes toward the alcoholic patient.
- 4. Underlining the importance of early diagnosis and a "high index of suspicion."
- 5. Briefly describing the process of treatment and the advantage of a "team approach" in caring for the alcoholic.

Definition

The American Medical Association has defined alcoholism as "an illness characterized by significant impairment that is directly associated with persistent and excessive use of alcohol. Impairment may involve physiological, psychological or social dysfunction." Such a diagnostic approach does not require that the cli-

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nician focus on the pattern of consumption, the quantity consumed, or the type of alcoholic beverage preferred (information which is often difficult to verify). Instead only evidence of significant medical, occupational, family, marital, legal, emotional, and financial consequences of alcohol use is necessary to establish the diagnosis. Certainly, individuals not suffering from alcoholism may experience an alcohol-related difficulty. Such a person, however, is capable of successfully modifying his drinking behavior to avoid future problems. The hallmark of the alcoholic person is persistent drinking in the face of obvious alcohol-related consequences. As is the case with other chronic diseases such as hypertension, early in the course of alcoholism it is occasionally difficult to separate alcoholies from non-alcoholies.

Alcoholism as a Disease

The acceptance of alcoholism as a medical disorder is a relatively recent development. Although Benjamin Rush characterized alcoholism as a disease in the mid-19th century, the "disease concept" lost favor until the work of E.M. Jellinek in the 1950s began to produce evidence for a medical model of alcoholism. In the 1970s Goodwin clearly demonstrated an important genetic contribution in the etiology of the disease. His studies revealed that individuals with a biologic alcoholic parent have a fourfold increased risk of developing alcoholism, even when adopted and raised by non-alcoholic parents. In addition, four separate twin studies have shown a significantly greater concordance for alcohol abuse between monozygotic twins when compared with dizgotic twins.

In 1983 George Vaillant published his monumental investigation of a large non-alcoholic population which was meticulously characterized for numerous psychosocial variables and followed for 40 years. Because of the longitudinal, prospective design, he

was able to identify those individuals who developed alcoholism and then determine if there were any consistent premorbid traits or predictors of subsequent alcoholism. He found that the so-called "alcoholic personality" was a result of an individual's alcoholism and did not reflect an underlying personality type or disorder which predisposes to the development of alcoholism.6 In addition to genetic factors Vaillant found that cultures which "introduce children to the ceremonial and sanctioned use of low-proof alcoholic beverages taken with meals in the presence of others, coupled with social sanctions against drunkeness" have less alcoholism.

In short, we have come to see alcoholism as a multifactorial disease whose determinants in any specific individual may be differently weighted, not unlike hypertension or diabetes. Therefore, alcoholism, like hypertension, is "essential" in the majority of cases, and is less frequently seen as a symptom or expression of an underlying psychiatric or personality disorder. It is true that many alcoholics are depressed, but typically their depression is a result, not a cause, of their alcoholism.

Finally and perhaps most persuasively, viewing alcoholism as a disease is worthwhile because it provides the foundation for the beginning of recovery itself. It offers a rational explanation to the patient for his own poorly understood, erratic, and destructive behavior and allows the patient to deal with guilt which is already excessive and often perpetuates or exacerbates his addiction. And contrary to the objections of some, it makes the patient more, not less, responsible for his own recovery. By making the diagnosis of alcoholism we are in a position of showing the patient how to assume responsibility for his disease.

Physician Attitudes

Physician attitudes have been long recognized as important determi-

nants of treatment outcome. In years past, before the proliferation of highly technical diagnostic tests and the vast array of therapeutic modalities, the physician possessed a limited armamentarium for diagnosis and treatment. At the turn of the century William Osler, chairman of the Department of Medicine at Johns Hopkins, was well known as a great "healer." This intangible ability was attributed by his associates and staff to his behavior and attitude and was felt to be independent of his vast knowledge of medicine. He credited his results to "Faith in Saint Johns Hopkins, as we used to call him, an atmosphere of optimism, and cheerful nurses."8

Physician attitudes and behavior can also work in negative ways. Fisher showed that students he studied in the second year of medical school demonstrated significantly negative and pessimistic attitudes toward alcoholics. Their view of the alcoholic patient became progressively more negative during the remainder of their education, especially during residency training.9 Housestaff in large urban hospitals often encounter the indigent, end-stage alcoholic with few remaining support systems. Patients in the end stages of any disease generally have the poorest prognosis and alcoholies are no exception.

Young physicians, unfortunately, carry this perception of alcoholism with them as they enter practice where the "down and out" alcoholic represents less than 5% of the total alcoholic population. The result is underdiagnosis of alcoholism and cynicism concerning treatment. The most functional and salvageable patients escape identification and the "self fulfilling prophecy" is perpetuated. It is understandably difficult for physicians to divorce themselves from the pervasive, culturally determined attitudes about alcohol in our society. These attitudes include such notions as: "excessive drinking is a sin," "alcoholies are weak," and "aleoholies

drink to cover up character defects or personal inadequacies." It is these same attitudes which make it difficult for the alcoholic patient to admit his problem.

Diagnostic Clues

Over half of alcoholics seen by physicians go undiagnosed. Recently. however, awareness is increasing that identification and treatment of patients in the early stages of alcoholism can result in reversal of their psychosocial and medical problems even though treatment cannot be viewed as a "cure." In the early stages of their illness alcoholics frequently come to medical attention but not usually to seek help for alcoholism or to discuss the consequences of their drinking. They typically seek treatment for related problems: gastroincomplaints, insomnia, depression, anxiety, or nervousness. Major and minor trauma are also common manifestations of "early alcoholism." Because these problems are not specific for alcoholism, and there are no biochemical markers specific for alcoholism, a considerable degree of suspicion for the possibility of underlying alcoholism must be maintained in order to make the diagnosis.

When alcoholism is suspected, obtaining a more detailed drinking history is advised. Alcoholics often skillfully hide and protect their drinking not because they are basically dishonest or because they like to aggravate their physicians (as it may seem), but because this behavior is a manifestation or expected consequence of the disease itself. They are often fearful and guilt ridden about their drinking and its consequences and cannot be expected to openly reveal their symptoms and problems. They recognize the social stigma associated with the "alcoholic" label. A number of questionnaires have been developed to assist the physician in taking the history (for example, the Michigan Alcoholism Screening Test).10 Historical data can also be

TABLE 1

Disease States Which Frequently Complicate Alcoholism Acute and chronic pancreatitis Gastritis Gastric and duodenal erosions or ulcerations Esophagitis Mallory Weiss Syndrome Fatty liver, hepatitis, cirrhosis Diarrhea, malabsorption Cardiomyopathy Hypertension Fractures, Contusions, subdural hematomas, and other trauma Anemia, thrombocytopenia, leuko-Impotence and hypogonadism Gout Ketoacidosis Nutritional Deficiencies Dementia Polyneuropathy Cerebellar ataxia Wernicke Korsahoff Syndrome Myopathy Malignancies of oral pharynx and esophagus Intoxication and withdrawal syn-

verified by talking with family members and others close to the patient. It is essential that the interview be conducted in a non-moralistic and non-judgmental tone.

dromes

Additional clinical and laboratory information may help alert the physician to the possibility of "occult" alcoholism. Patients who present with diseases known to frequently result from alcoholism need an aggressive approach to the diagnosis and treatment of their alcohol dependence (see *Table 1*).

A variety of metabolic derangements and abnormalities on routine chemistry profiles are often seen in alcoholic patients which may increase the physician's suspicion as well as assist him in confronting the patient (see *Table 2*). Many alcoholics have entirely normal laboratory data but several abnormalities are so common

that their presence should prompt the physician to consider alcoholism as a differential diagnostic possibility. These studies include mean corpuscular volume (MCV), the gamma glutamyl transpeptidase (GGTP), routine liver enzymes, and blood or breath alcohol determinations.

The mean red blood cell volume (MCV) is often increased in alcoholics and may be the result of folate deficiency, vitamin B12 deficiency (less common), liver disease, or reticulocytosis. In one study of alcoholics consuming greater than 80 grams of ethanol daily (8 oz. of 40 proof beverage equivalent), 89% of patients were noted to have macrocytosis.12 Patients who drink in an episodic or "binge" pattern will less frequently have macrocytic changes. In another study of an alcohol treatment center population, 44% of the patients had a MCV greater than 100 and/or macrocytic changes on peripheral blood smear.13 The MCV, therefore, can serve as a useful screening device especially because of its wide availability on coulter blood counts.

Gamma glutamyl transpeptidase (GGTP), a hepatic microsomal enzyme, is generally a more sensitive indicator of alcoholic liver injury than the SGOT or SGPT. It is reportedly increased in 50-90% of chronic heavy drinkers. It is also often elevated in patients with nonalcoholic liver diver disease, obstructive biliary tract disease, and may be elevated in patients on chronic drug therapy known to induce hepatic microsomal enzymes such as phenobarbital and Dilantin. If its limitations are recognized it also can be quite useful.

Hepatic transminase values (SGOT-SGPT) are often elevated in alcoholic liver disease. In alcoholic hepatitis, the SGOT is classically higher than the SGPT, and in many cases of alcoholic liver disease the elevations are relatively low grade (compared with typical viral or toxic liver injury). Therefore, even minimal SGOT and SGPT elevations should not be

ignored and may serve to increase suspicion of alcoholism.

In one study, Ryback showed that a statistical or computer analysis of several routine automated laboratory tests could be used to accurately screen for alcoholism.¹⁵ His method correctly identified 100% of alcoholic patients on a medical ward, 94% of alcoholics in an alcoholism treatment center, and 100% of a control group of non-alcoholics.

By keeping in mind the common ways alcoholics present to physicians, the disease states for which alcoholics are at risk, and the simple laboratory tests which can serve to arouse suspicion and lead to identification, the clinician can markedly improve his ability to diagnose this common and generally progressive disease.

Treatment

Treatment begins by establishing the diagnosis of alcoholism and communicating this to the patient. This is best accomplished, not by informing the patient at the initial contact that he or she is alcoholic, but by helping the individual make the connection between his drinking and the problems which have occurred as a result. Alcoholic patients have a notorious difficulty making this connection on their own.

By reviewing with the patient any evidence of impairment related to alcohol (for example, marital discord, job absenteeism, a DWI charge) as well as the significance of abnormal laboratory tests, symptoms or physical findings related to the use of alcohol, the patient can be helped to overcome the natural tendency to rationalize, minimize, or deny the problem.

Chafetz suggests that "in handling the initial contact, consistent respect for the alcoholic's tenuous feelings of self esteem and constructive utilization of his dependency needs are essential. This means treating him with respect and consideration, reducing the frequency of frustrating situaTABLE 2

Endocrine and Metabolic Effects of Alcoholism

Alcoholic Ketoacidosis Decreased Testosterone Hypercholesterolemia Hyperglycemia Hyperlactatemia Hypermetabolism Hypertriglyceridemia Hyperuricemia Hypoglycemia Hypokalemia Hypomagnesemia Hypophosphatemia Hypotransferrinemia Metabolic Acidosis Protein Malnutrition Respiratory Acidosis Vitamin B Deficiencies

tions, and gratifying his requests."16

Many patients experience a sense of panic when they face the loss of their main coping mechanism even though they may recognize it as selfdestructive. In such cases, having the patient talk with someone who has successfully made the transition to abstinence, such as a member of AA, can be very useful. A reasonable goal to be achieved at the initial encounter with the patient might be for him to acknowledge that his use of alcohol is contributing to one or more problems and therefore he has an "alcohol problem." For patients resistant to this approach, a conference or "intervention" can be arranged with the patient's family and other significant individuals close to the problem. The participants in this meeting can supportively but firmly express their concern and perception of the patient's deterioration as a result of drinking. Such a meeting is best conducted by someone trained in alcoholism counseling.

The patient considering treatment should be informed that trying to "handle it" on his own is the least acceptable and typically the most unsuccessful strategy. Just as the overall care of diabetes has been improved by a "team approach," the treatment of alcoholism optimally involves the expertise of doctors, nurses, counselors, dieticians, and others with specialized training. The primary physician frequently does not have the time or expertise to provide these services and may want to refer the patient to an inpatient or outpatient alcohol treatment program where the comprehensive needs of the patient can be addressed and a treatment plan formulated.

The decision to utilize inpatient or outpatient services is dependent on several factors. If the patient is physically dependent on alcohol, an inpatient setting where the patient can be monitored and medications administered is recommended for most patients and mandatory for patients with a history of seizures or past withdrawal complications. Patients with serious medical problems such as alcoholic hepatitis, dehydration, and nutritional deficiencies also are best treated, at least initially, as inpatients. In addition, patients with few support systems, poor social environments for recovery, questionable motivation, or anticipated difficulty remaining drug or alcohol free in an outpatient program, are good candidates for inpatient treatment. Patients who are relatively healthy, appear motivated, and are not anticipated to have significant withdrawal may do well in an outpatient program.

For those patients requiring treatment of withdrawal, a gradually decreasing schedule of chlordizepoxide or lorazepam is generally prescribed and adjusted to the individual needs of the patient. Chlordiazepoxide (Librium) has a longer half life and possibly less potential to produce a subjective "high." Lorazepam (Ativan) has a short half life, relatively simple hepatic metabolism and is less likely to accumulate and cause oversedation in elderly patients or those with advanced liver disease. Detoxi-

fication is usually accomplished in five to seven days but may be prolonged in patients with polydrug dependency (especially involving benzodiazepines or other CNS depressants). The aim should be to provide comfort, but the medication should not be continued after the expected withdrawal period. Supplemental vitamins including thiamine and folate are given, fluids and nutrition are encouraged, vital signs are monitored, and other medical problems are identified and treated during detoxification.

The rehabilitation aspects of treatment usually include intensive education about alcoholism, group therapy, family therapy and education, individual counseling, stress reduction and relaxation techniques, and introduction to Alcoholics Anonymous (see Table 2). Physicians can hopefully supplement this process with their own encouragement and follow-up. The goal of treatment is always complete abstinence. For patients completing inpatient or outpatient treatment, an ongoing program designed to maintain sobriety is critical. For most patients AA is the heart of this program. AA is an enormously successful organization with 1-2 million successfully recovering members. It provides recovering patients with positive role models and a social setting where alcohol and drug use is discouraged. Members experience acceptance, approval, and encouragement from their fellow members in a program that is free and available worldwide.

Disulfiram (Antabuse) is also a useful adjunct in selected patients. It helps remove the temptation to drink during the initial stages of recovery. It is rarely successful by itself and patients must be encouraged from relying on it totally for maintenance of sobriety. It is relatively contraindicated in patients for whom an Antabuse reaction would be very poorly tolerated (patients with severe coronary artery disease, cerebrovascular disease, extremely severe liver dis-

ease, malignant hypertension, etc.). It should also be used very cautiously in patients with diabetes, hypothyroidism, epilepsy, or history of psychosis. In addition, it may inhibit the biotransformation of several drugs including the benzodiazepines, isoniazid, phenytoin, rifampin and warfarin.¹⁷

The course of alcoholism and the individual patterns of alcoholic drinking are extremely variable, a phenomenon Jellinek explained by describing several types of alcoholism as a unitary disorder with a broad spectrum of severity, natural history, and response to treatment. Alcoholism is a chronic disease, and like other chronic diseases, relapse is not uncommon. The pessimism for treatment prevalent among physicians in the past, however, is no longer justified.

Studies designed to determine the outcome of treatment have yielded a variety of results but have consistently indicated that patients experience significantly less psychosocial impairment following treatment. Vaillant reports that "half of all alcoholics achieve stable recoveries and a significant number achieve stable remissions the first time they seriously seek clinical treatment." Treating alcoholism has also been shown to result in decreased utilization of health care resources and less absenteeism from work.

Since family treatment is now a part of most rehabilitation programs, by treating alcoholics we frequently provide understanding and relief to family members and loved ones who have suffered emotional and sometimes physical trauma at the hands of the out-of-control alcoholic.

If physicians can reserve moral judgment, keep in mind the signs and symptoms of early alcoholism, and maintain an aggressive approach to the identification and treatment of their alcoholic patient, they will find that their efforts will usually meet with success and appreciation. The

diagnosis and treatment of alcoholism remain challenges worthy of the efforts of the medical profession.

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Limb Salvage Surgery for Severe Leg Ischemia

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Since the onset of the use of the saphenous vein for leg ischemia, the indications for its use have been gradually broadening. Originally, the vein graft was used as a femoral to popliteal graft in the region of the knee. Recently, the indications and vein grafts have been extended so that the distal anastomosis is occasionally done at the ankle or beyond to effect limb salvage.

Presentation of a Case

The patient is a 72-year-old diabetic who developed ischemic rest pain in his right foot in April 1984. An aortogram done at another hospital showed patent aorto-iliac segments with obstructive disease in the superficial femoral, popliteal and its trifurcation. The films showed no patent distal vessel for anastomosis but the films terminated four inches above the ankle. The patient was deemed inoperable and had a right lumbar sympathectomy. Postoperatively, the patient had no relief of his rest pain and required narcotics for the pain.

The patient was admitted to Methodist Hospital for evaluation in July 1984. After preliminary workup was completed, the patient was taken to

surgery and an operative arteriogram (Figure) showed a segment of anterior tibial artery near the ankle was patent. A nonreversed vein graft from the common femoral to the anterior tibial artery at the ankle was performed.

Postoperatively, the patient maintained a good dorsalis pedal pulse and his foot was asymptomatic.

Discussion

With increasing experience in the use of the saphenous vein graft, longer vein grafts to smaller vessels with poorer run-off have been attempted.

In patients with severe ischemic rest pain, ischemic ulcers of the foot or gangrene of the toes, surgeons have taken vein bypasses to the ankle and beyond, if suitable arteries could be identified.

We have performed 18 consecutive limb salvage attempts using the saphenous vein as the graft material to vessels in the leg distal to the popliteal artery. Ten of these patients were diabetic. There were nine women and nine men. Average age for men was 67 years and for the women the average was 72 years. Two patients went on to below-knee amputation in spite of our attempts. In one of these (a diabetic), deep ulcerative gangrene of the foot progressed in spite of a patent graft to the distal anterior tibial artery. In two patients, routine abdominal aortography with run-off failed to show any vessels near the ankle, but arteriograms in surgery revealed patent vessels near the ankle that accepted a graft. Two additional patients had no Doppler signals at the ankle or foot but were subsequently found to have vessels to attach grafts to.

Three patients with gangrene of a toe had successful amputation of that

toe during the same hospitalization with healing. There were no hospital deaths or complications in this group during their hospitalization.

Only one patient was not immediately improved following surgery. He was our first patient in this series and the distal graft was to a blind segment of the posterior tibial artery. He was one of the two patients who went on to eventual amputation (three months later).

Two patients occluded their vein grafts. One of these was the gentleman just described, and another was a 76-year-old woman with a one-inch ischemic ulcer of the medial malleolus who had a graft to her posterior tibial artery an inch from the ulcer. Her vein graft remained open 4.5 months before it closed, long enough to heal her ischemic ulcer. She has thus far avoided an amputation.

In each of these patients, we were able to use the entire saphenous vein for grafting. We turned the graft inside out and resected the valves and returned the vein to its original position. This allowed us to use the entire vein and anastomose the smallest part of the vein to the small branch artery as indicated.

This technique, previously described, allowed us to use the entire saphenous vein or portion of it in each patient. In two patients, we had to go to the opposite leg to obtain either the entire saphenous vein or a portion of it.

Summary

Eighteen consecutive patients facing leg amputation because of ischemic rest pain, ischemic ulcers or gangrene of the toes were submitted to nonreversed saphenous vein bypass surgery. All but one patient was

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improved during that hospitalization. Since then, that patient and one other have undergone below-knee leg amputations (the second patient in spite of a patent vein graft). One other patient has clotted her graft more than four months after surgery, after her ischemic ulcer had healed and she has thus far avoided amputation.

Although the long-term results in this group of elderly patients awaits evaluation, the initial impression remains that aggressive revascularization as far as the ankle should be considered in patients threatened with leg amputation.

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AN OPERATIVE ARTERIOGRAM on a patient pronounced inoperable shows (arrow) an anterior tibial artery suitable for grafting.

PATIENTS NEEDED FOR DIABETES RESEARCH STUDY

The Diabetes Research and Training Center, Indiana University School of Medicine, is seeking patients for studies with an experimental aldose reductase inhibitor to determine if this drug will prevent or retard the development of diabetic retinopathy. Aldose reductase inhibitors work by preventing the accumulation of sorbital in tissues including the lens, nerves and retina. They have already been found to prevent metabolic cataracts and to improve diabetic neuropathy in experimental trials. Since the retinal cells accumulate sorbital, this trial is designed to determine if the administration of the drug would prevent retinopathy or retard its progress. The drug is ex-

perimental and although no serious side effects have been found, this study will require close follow-up for one to two years. The potential benefit for patients would be close follow-up of their diabetes and retinopathy including retinal photographs and the possible prevention of a serious and disabling complication of diabetes. The trial is in a double-blind format. Otherwise healthy patients with either type I (juvenile) or type II (adult) diabetes are being sought. Patients either without retinopathy or with non-proliferative retinopathy would qualify. Patient referral may be made by calling or having the patients call the Diabetes Center at (317) 630-6374.

CEA Immunoperoxidase Staining

An Applied Research Tool for a Community Hospital Laboratory

TIMOTHY T. DICK, M.A., c(ASCP) CARLENE E. WOLF, HT (ASCP) PRAMOD K. CARPENTER, M.D. Evansville

Immunoperoxidase
Staining Is Becoming
an Effective Method
for Assisting with
the Diagnosis and
Classification of
Malignancies . . .

OMMERCIALLY AVAILABLE immunoperoxidase kits for the detection of a variety of cell antigens are currently available to the community hospital laboratory. Immunoperoxidase involves the localization of a specific antigen via a primary antibody with subsequent binding by a peroxidase anti-peroxidase bridge. Visualization of binding occurs following application of a chromogen such as diaminobenzidine or 3-amino-9-ethyl carbozole. Of some interest has been the use of carcinoembryonic antigen (CEA) identification in tissue to aid in the typing of malignancy. Recent studies have evaluated the presence of CEA in tissues of various malignancies, in particular, colorectal,7.9 and small cell carcinoma of the lung.10,11 In an attempt to demonstrate the applicability of immunoperoxidase staining for CEA and its use by a 600-bed community hospital, 32 primary tumors were immunohistochemically stained for CEA.

Materials and Methods

Surgery patients diagnosed as having had one of 32 primary malignancies for the year 1983 were supplied by the Deaconess Hospital Tumor Registry. Sections from formalin-fixed, paraffin-embedded tissue were obtained with subsequent staining for CEA according to the manufacturer's directions (Histoset, Ortho Pharmaceutical Co., Raritan, N.J.).

Results

Table 1 depicts the results of immunoperoxidase CEA staining for 32 sites of primary malignancy (mixed

histopathology). Table 2 lists the sites which stained positive, and shows the total number of sections examined.

Discussion

Many community hospital laboratories in Indiana have limited research capacities and resources. This study demonstrates that commercially prepared immunoperoxidase kits can be effectively utilized by these laboratories for general study and specific application. Tables 1 and 2 show that at least one section of appendix, cervix, colon, lung (nonsmall cell), pancreas, rectum, small intestine and stomach stained positive for CEA, agreeing with previous reports.3.5,6 In addition, none of the following tissues (brain, breast, esophagus, prostate and skin) stained for CEA, concurring with other studies.3.5.6 Cases previously reported to have stained for CEA, lung (small cell) and ovary, were not successfully stained during the course of this study. However, occasional negative results are to be expected.

Despite the sensitivity of the immunoperoxidase techniques, preservation of antigenicity requires attention to the type of fixative and temperature control during processing and avoidance of undue delay in sectioning and staining. Bone, hypopharynx, lip, melanoma, salivary gland, testicular, thyroid, tongue and two sections of uterine tissue were found to stain negatively for CEA, whereas, nasal cavity and oropharynx were found to stain positively. Limited information is available concerning CEA staining for most of these tissues. Of particular note, gallblad-

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		7	ΓΑΒ	LE 1	
Staining	Pattern	of	AH	Malignancies	Evaluated

SITE	HISTOPATHOLOGY	CEA + / -	SITE	HISTOPATHOLOGY	CEA + I -
Appendix	Adenocarcinoma	weakly +	Lung-SC	Oat Cell	
	Carcinoid	_	1	Oat Cell	
Bladder	Transitional Cell	-	Melanoma	Urinary bladder	-
	Papillary Trans. Cell			Polypoid, right leg	
	Transitional Cell		Nasal Cavity	Squamous & Trans. Cell	weakly +
Bone	Chondrosarcoma			Transitional Cell	www
	Chondrosarcoma	-	Nasopharynx	Squamous Cell	***
	Chondrosarcoma		Oropharynx	Squamous Cell	+
Brain	Astrocytoma		Ovary	Adenocarcinoma	
	Oligodendroglioma		Pancreas	Adenocarcinoma	_
	Meningioma			Adenocarcinoma	+ + +
Breast	Adenocarcinoma			Adenocarcinoma	
	Adenocarcinoma	white	Prostate	Adenocarcinoma	
Cervix	Squamous	weakly +		Adenocarcinoma	
	Adenocarcinoma	_	Rectum	Adenocarcinoma	
	Squamous	+ +		Adenocarcinoma	+ +
Colon	Adenocarcinoma	+ +		Adenocarcinoma	+ + +
	Adenocarcinoma	+ + +	Salivary Gland	Adenocarcinoma	
	Adenocarcinoma	+ + +	Skin	Squamous Cell	
Esophagus	Squamous cell	-		Squamous Cell	_
	Adenocarcinoma	-		Squamous Cell	
Gallbladder	Adenocarcinoma	weakly +	Small Intestine	Carcinoid	+ +
	Adenocarcinoma		Stomach	Adenocarcinoma	+
Hypopharynx	Squamous cell	-		Adenocarcinoma	+ +
Kidney	Renal Cell Carcinoma			Adenocarcinoma	+ + +
	Transitional Cell		Testicular	Seminoma	_
Larynx	Squamous Cell			Seminoma	_
	Squamous Cell	-		Teratoma	
	Squamous Cell		Thyroid	Papillary Follicular	
Lip	Squamous Cell			Carcinoma	
Liver	Hepatocarcinoma	+	Tongue	Squamous Cell	
Lung-NSC	Adenocarcinoma	++	Uterus	Adenocarcinoma	
	Adenocarcinoma	+ +		Hydatiform mole	
	Adenocarcinoma	+			

TABLE 2 Number of Tissues Staining Positive for CEA Compared with Total Number Tested

SITE	NUMBER TESTED	NUMBER POSITIVE FOR CEA
Appendix	2	1
Breast	2	1
Cervix	3	2
Colon	3	3
Gallbladder	2	1
liver	1	1
ung (non-small cell)	3	3
Jasal Cavity	1	1
Propharynx	1	1
ancreas	3	1
Rectum	3	2
Small Intestine	1	1
Stomach	3	3

der and liver stained for CEA during this study but had been reported as negative by Goldenberg, *et al.*³⁶ Additional staining of gallbladder and liver tumors is required to substantiate these findings.

Immunoperoxidase staining is becoming an effective method for assisting with diagnosis and classification of malignancies. This study has shown that community hospitals can utilize immunoperoxidase procedures for applied research and as an enhancement of routine methods now in use.

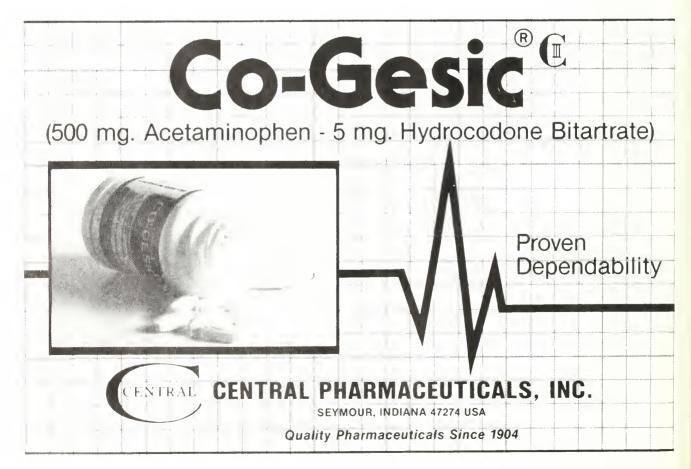
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Carcinoma of the Breast

Identical Twins Differing in Time of Onset and Severity Implicate Non-Genetic Factors Influencing Clinical Course

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F ALL THE CANCERS in women breast cancer has been the subject of extensive genetic studies.142 The high incidence of breast cancer occurring in first degree relatives of women is considered to be due to genetic factors.3 In this paper we report carcinoma of the breast in a pair of identical twins with markedly different age of onset, site of metastasis, clinical course and survival. Any difference in identical twins must be environmental in origin since they are genetically identical. If environmental factors significantly affect the course of breast cancer, their characterization may lead to more effective treatment.

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Abstract

A pair of identical twins concordant for carcinoma of the breast were studied to explain their clinical differences.

The less severely affected twin had pre-menopausal occurrence of breast cancer, late pre-menopausal oopher-ectomy, metastasis occurring after 24 years of disease free interval and she is alive.

The more severely affected had early pre-menopausal oopherectomy,

late menopausal occurrence of breast cancer with a negative estrogen receptor status and she died with rapid development of metastasis.

We conclude that in breast cancer: 1. Genotype alone does not determine clinical course. 2. Pre-menopausal oopherectomy can probably delay the occurrence or metastatic behavior. 3. Estrogen receptor status is probably determined by environmental factors and indicates poor prognosis when negative.

Case Reports

Twin I was first-born (1918). She was diagnosed to have carcinoma of the breast at age 40. She underwent mastectomy on the left side with postoperative radiotherapy and no further treatment. She underwent hysterectomy with bilateral oopherectomy at age 43 for menstrual problems only. At age 64, in March 1983, she developed abdominal pain and a laparotomy showed pelvic mass consistent with metastatic carcinoma of the breast by histopathology. No other metastasis could be documented by radiographic studies. She received megavoltage radiotherapy and remains asymptomatic now (1984). She attained menarche at age 13 and had first childbirth at age 25 and lived in Michigan since age 24.

Twin II, the first patient's identical sister, presented with a large lump in the right breast at age 64, April 1982. She underwent a modified radical mastectomy and she received various combinations of chemotherapy, postoperatively, despite which she developed lung metastasis by June 1983 and extensive liver metastasis by November 1983. She died in December 1983 with extensive bilateral pulmonary infiltrates suggestive of lymphangitic involvement with metastatic carcinoma of the breast. In addition, an autopsy revealed micrometastasis of cerebral hemispheres. There was no evidence of bone metastasis! She attained menarche at age 13 just like her twin sister, her first childbirth at age 22. She also had hysterectomy and bilateral oopherectomy for menstrual problems but much earlier, at age 31. She lived in Indiana since age 19.

The family history of both twins is significant; their mother died of breast cancer at age 56 and their father died of carcinoma of the prostate.

Results

The twins were probably monozygotic in view of the marked resemblance in the recent photograph (Fig. 1) and in childhood (Fig. 2). The probability of monozygocity was further confirmed by comparison of blood group systems by the method of Maynard-Smith and Penrose.4 The histopathology report of both twins showed duct cell carcinoma of the breast. Twin I had simple mastectomy but Twin II had modified radical mastectomy showing 10 of 12 axillary lymph nodes involved with extensive perivascular invasion. No steroid receptor studies were conducted in Twin I. The estrogen and progesterone receptors were negative in Twin II. Fig. 3 compares the clinical course of twins from birth until 1984 and the Table compares their clinical differences.

Discussion

We know little about the actual cause of breast cancer but we know a great deal about the risk factors defined as the characteristics of individual patients that increase the chances of developing breast cancer above the level of risk for the general population. 5.6 These risk factors may be generally classified as genetic (family history), hormonal (age at menarche, menopause and first childbirth), nutritional (socioeconomic, ethnic factors) and radiation. Analysis of these risk factors by comparison in these identical twins may help to determine the significance in the causation of breast cancer.

The high frequency of breast cancer occurring among first degree relatives of women is said to be due to genetic factors. Since the twins' mother had breast cancer, its occurrence in these identical twins is not unique. However, carcinoma of the breast in identical twins has seldom been reported^{7,8,9} and even the data from Danish Twin Registry fails to answer the genetic or environmental factors influencing the clinical course.¹⁰

Early menarche is considered to be



FIGURE 1: Identical twins at age 64. Twin I (left) had mastectomy 25 years ago. Twin II (right) had mastectomy in 1982.



FIGURE 2: Twin I and Twin II in childhood.

one of the risk factors for occurrence of breast cancer. Since both twins attained menarche at normal age and at the same time, age at menarche was not a significant factor in the occurrence of their breast cancer. Early first pregnancy appears to be associated with reduced risk by making the breast tissue less susceptible

for carcinogenic stimulus. Twin I had first childbirth at age 25, Twin II at age 22. The age difference, though not significant for the general population, could have offered some protection to Twin II from early breast cancer compared to her identical twin.

Ovarian activity in women appears to play a significant role in the inci-

dence of breast cancer. Pre-menopausal oopherectomy reduces the risk and degree of protection and is inversely related to the age at which the ovaries were removed. Women with surgical menopause had only 63% risk of breast cancer of women who had natural menopause between the ages of 45 and 54. The reduction in risk was greatest among women with surgical menopause before age 35

The most important difference in the clinical course of these twins appears to be the age at which they had attained surgical menopause and age at which the breast cancer occurred. Twin I was still in pre-menopausal state while developing breast

caneer at age 40. Twin II was probably protected from developing the cancer at the same time due to surgical oopherectomy several years before. It is also important to note that Twin I developed metastasis of breast cancer in post-menopausal age of 64 despite her bilateral oopherectomy at a pre-menopausal age of 43 and Twin II developed primary breast cancer at age 64 despite her bilateral oopherectomy at pre-menopausal age of 31. While pre-menopausal oopherectomy could be protecting women from occurrence or metastasis of breast cancer prior to natural menopausal age, it appears to have no bearing on the development of primary or metastatic carcinoma of the breast at late post-menopausal age.

Despite the differences in pre-menopausal age, the biology of breast cancer in these twins appears to converge suddenly at one point, at age 65, with development of metastasis in Twin I and Twin II almost simultaneously. This may suggest that while environmental risk factors are important in the occurrence of breast cancer, genetic influences may have a more important role in the development of metastasis. This may also explain the predilection for soft tissue metastasis in these twins and surprisingly sparing them both from bone metastasis, which is otherwise a common event in the clinical course of metastatic carcinoma of the breast.

Steroid hormone receptors in breast cancer have been shown to be good indicators for prognosis.14 Patients with positive estrogen receptors generally do well with prolonged diseasefree survival as compared to estrogen receptor negative patients who are known to progress rapidly with visceral metastasis. Since Twin I showed good prognosis with prolonged disease-free interval and responded well to treatment of her metastasis, one can probably assume safely that she was estrogen receptor positive although these studies were not conducted. Twin II was negative for steroid receptors and her prognosis proved to be very poor with rapid development of visceral metastasis and death. This observation suggests that steroid receptor status is probably determined by environmental factors.

There is geographic variation in the incidence of breast cancer worldwide and studies have shown high incidence in lower temperature zones. It is interesting to note that Twin I, who developed breast cancer earlier, lived in the colder part of Michigan whereas Twin II, who developed her cancer several years later, lived in Indiana with a much warmer climate.

It is becoming clear that the key to understanding cancer causes might be found in those genes collectively

19	88	- 70		
			Alive Pelvic Metastasis	Died - Liver, Lung Metastasis CANCER. Right Mastectomy,
19	78	- 60		Duct Cell Carcinoma
19	68	- 50		
			SURGICAL MENOPAUSE	
19	58	- 40	CANCER. Left Mastectomy, Duct Cell Carcinoma	
	4.0	- 30	Second Childbirth	SURGICAL MENOPAUSE
19	40	- 30		
			First Childbirth	
19	38	- 20	Married	First Childbirth Married
19	28		Menarche	Menarche
19	18	Birth	Birth	Birth
	\dashv			
YE	AR	AGE	TWINI	TWIN II
1				

FIGURE 3: Clinical course.

termed oneogenes.10 Evidence now indicates that cellular oncogene activation is an important step in the carcinogenesis. Oncogenes from breast caneer have been isolated recently.16 Whether participation of oncogenes is required early in the formation of cancer or during its progress is not yet known. It is possible that there are multiple sets of oncogenes involved from the occurrence to the progression of a cancer, different sets of oncogenes activated at each step. 17 Since these twins with breast cancer were genetically identical, they probably shared the same oncogenes for breast cancer. Marked clinical differences in their pre-menopausal and post-menopausal age suggest different sets of oncogenes activation. It is possible that one set of oncogene activation took place in Twin I at pre-menopausal age due to intact ovarian function and the oncogenes in Twin II were not activated at the same time due to oopherectomy. The simultaneous occurrence of metastasis in these twins in postmenopausal age suggests different sets of oncogene activation.

Conclusion

We are not aware of any previous reports of identical twins with breast cancer differing in clinical course. From our study of these twins we conclude that in breast cancer: 1. Genotype alone does not determine the clinical course. 2. Intact ovarian function prior to natural menopause puts the patient at higher risk for occurrence of breast cancer when she is susceptible for transformation of a set of oncogenes by environmental factors, whereas surgical oopherectomy at early menopause probably protects a woman. 3. Pre-menopausal oopherectomy conducted early or late in pre-menopause before or after occurrence of breast cancer probably has no influence in the clinical course of breast cancer at post-menopause. 4. Steroid receptor status for breast cancer is probably determined by en-

	TABLE Clinical Differences	
	Twin I	Twin II
First Pregnancy	Age 25	Age 22
Second Pregnancy	Age 32	None
Diagnosis of Cancer	Age 40 (1958)	Age 64 (1982)
Site	Left Breast	Right Breast
Steroid Receptor Status	Unknown	ER, PR Negative
Metastasis	25 Years After	1 Year After
	Diagnosis of	Diagnosis of
	Primary (1983)	Primary (1983)
	Pelvie	Liver, Lung and
	Metastasis	Brain Metastasis
Iormonal Constitution at	27 Years of	33 Years of
Time of Diagnosis	Pre-Menopause	Surgical
	·	Post-Menopause
leographic Regions Lived	14 Years in	32 Years in
at Time of Diagnosis	Michigan	Indiana
urvival	Alive with	Died with Liver,
	Pelvic	Lung and Brain
	Metastasis	Metastasis

vironmental factors rather than genetic. 5. Different sets of oncogenes are probably activated pre-menopausal and post-menopausal breast cancer in its occurrence and metastasis.

We are not supposing that the difference in courses is entirely explained by differences found in our investigation. However, any difference in course must be due to environmental factors in these identical twins. We hope this report will stimulate others to report clinically identical twins with breast cancer in the hope of illuminating environmental determinates which may lead to more effective methods of treatment.

Addendum

Twin I is alive with pelvic metastasis in clinical remission.

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Beta-Adrenergic Blocking Agents

Effect of Chronic Dosage on a Fit, Middle-Aged Man: A Case Study and Review

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Abstract

Evidence from the literature is reviewed for reduced utilization of muscle glycogen and reduced lipolysis resulting from beta-adrenergic blockade. Observation from the performances and from stress tests on a middle-aged athlete before and during his treatment with Tenormin (atenolol) and Trandate (lubetalol) are interpreted in terms of reduced substrate availability for muscular work.

ETA-ADRENERGIC BLOCKING DRUGS are commonly used to treat essential hypertension [1,2,3]. Exercise programs are also considered to be effective in reducing cardiovascular and pulmonary disease [4-9]. The physiological and biochemical changes that result from beta-adrenergic blockade seem to provide serious conflicts with a parallel exercise program whether for recreation, rehabilitation or competition. Most studies involving exercise testing on human subjects have been performed on young subjects and usually have employed acute administration of beta-blockers [13,18,20,21,23-30], often by a single dose. Only a few studies have been reported on the effects of chronic beta-blockade for young subjects [10,22], for older subjects [18], or for patients [31,32,35,36]. Although studies using acute dosage are obviously much easier to manage, it is not absolutely certain that all the observations so obtained are applicable to chronic dosage schedules. Therefore, the presence of moderate hypertension and its treatment with beta-blockers in a highly fit, middle-aged member of an exercise science research group provided an opportunity for some unique observations.

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Background

There are several different betaadrenergic blocking agents available for the treatment of hypertension. The prototype of these is Inderal (propranolol), which is nonspecific in its beta-adrenergic blocking functions. Others are more specific as either beta-1 or beta-2 blockers. In addition to the blood pressure lowering, each of these drugs may have other effects on the cardiovascular system and on carbohydrate and lipid metabolism. Each of these three effects may be a combination of several sites of action, and the extent of each of the three effects will vary considerably from one beta-blocker to another.

- 1. Blood pressure lowering will be the only desirable effect as far as the hypertensive patient is concerned.
- 2. The different beta-blockers vary considerably in the amount of lowering of heart rate and decreasing of cardiac output that they produce. This may be a serious problem for patients who have impaired cardiac function, or who have little cardiac reserve. On the other hand, this may present no problem to others. Indeed, weight lifters or body builders, for example, may be able to perform their training without difficulty.
- 3. The reduced carbohydrate and lipid metabolism in the muscle may be the least known of the side effects, and may not be noticed if the patient is not physically active. Hypoglycemia seems to accompany exercise, and is a distinct possibility even without physical activity.

Although the beta-blockers act quickly and are excreted rapidly [27] (more quickly by i.v. than by oral administration [30]), the full effects may require as much as three weeks [19]. From studies of both chronic and acute dosage, the beta-blockers have

been shown to produce a lowered heart rate [1,2,21,25,26,36] and lowered blood pressure [1,2,25,31,36] at rest and during exercise [31]. Changes in the peripheral circulation can be observed [26]. Maximum strength or the ability to do work lasting only a few seconds is unchanged [21,25]. Oxvgen consumption and carbon dioxide production during exercise are usually depressed [28]. During exercise the most obvious biochemical effects are the lowered ability to use heart and skeletal muscle glycogen [15,16,23] and free fatty acids from adipose tissues [14,16,23] as sources of fuel. As a consequence more blood glucose obtained from liver glycogen is used for muscle energy [14]. Although heart and skeletal muscle glycogen is thereby spared during exercise [15.16,34], hypoglycemia is a serious possibility [10,14] and produces a severe limitation to any but very shortterm exercise. Another consequence of the reduced utilization of muscle glycogen is that less lactic acid is being produced by means of anaerobic glycolysis and therefore lower lactate levels are found in the blood [10,14,16,21,23,35,36]. Possibly lactate utilization is also reduced, due in part to decreased splanchnic-hepatic blood flow [36].

During prolonged exercise, muscle glycogen is spared by the use of free fatty acids mobilized from adipose tissue [34]. This mobilization is diminished by the blocking of beta-1 receptors in adipose tissue [10-12,16,23] and as well by reduced blood flow in adipose tissue [13]. There is no evidence that there is any enhancement of cholesterol metabolism during exercise [33].

Changes in the density of beta-adrenoceptor density with respect to fitness have been found [17]. Differences in the effects of beta-blockade have been observed depending on the muscle type; performance of slow-twitch muscle seems to be more impaired [15,24]. It has been suggested that hypertensive subjects and nor-

TABLE 1								
Date	Event	Average BP	Resting pulse					
Jun 29, 1981	Test 1	not recorded	58					
Nov 19, 1981	Test 2	not recorded	58					
May 18, 1982	Test 3	160/110	56					
May 21-June 15	Tenormin 10mg/day	120/80	42					
July 6	1500 m in 5:18	140/85	54					
July 12	1500 m in 5:00	140/85	54					
Aug 15	1500 m in 4:57	150/100	54					
Aug 22-Sept 11	Tenormin 10 mg/day	135/85	48					
Sept 18	5000 m in 19:08	145/95	52					
Sept 21	Test 4	150/98	52					
Oct 19	Test 5	160/100	56					
Oct 19-Dec 2	Tenormin 20 mg/day	140/85	52					
Dec 2	Test 6	140/85	52					
Oct 1, 1983	400 meters in 58.3	150/95	55					
Sept-Oct 11, 1984	Trandate 400 mg/day	160/100	58					
Oct 11, 1984	Test 7	140/95	58					

motensive subjects respond differently to beta-blockade [20]. It has also been suggested that subjects cannot respond to training and conditioning while on beta-blockade [22].

The abrupt rise in circulating potassium during exhaustive exercise and the equally quick reversal upon completion of exercise has been well documented [37,38]. It has been shown directly by electron microprobe analysis [41] that muscle cells lose potassium during exercise. There have been conflicting reports as to whether or not certain of the beta-blockers interfere with these processes [39,40].

Subject

The subject was a 52-year-old man of middle European descent, 6 ft 2 in tall, weighing 165 to 170 lbs during the series of observations. The subject had been a 49 sec quarter-miler in college and had continued a regular exercise program. He had returned to serious training for Masters Track and Field competition in 1979. The subject usually trained five days each week for a total of 25 to 35 miles per week. At least half of this was interval or up-hill running. The subject had experienced a slow increase in blood pressure during the last 15 years. During 1982 he was treated with Tenormin (atenolol), a beta-1 blocker, and with Trandate (lubetalol) during 1984, while continuing to train, thus offering the opportunity to document the events presented in *Table 1*. The subject was of the opinion that his best conditioning occurred during the time of Test #4, that for all other tests being about equal. It may be noted that the subject's best 1500 meter time would have ranked about 30th in the U.S. for men, age group 50-54 and his 400 meter time about 17th [42].

Procedures

Stress tests were conducted at the Methodist Hospital Pulmonary Testing Laboratory. The subject exercised on an electrically braked ergocycle (Ergopneumotest, Wuerzburg, W. Germany). Five minutes of no-load pedaling were followed by 30 watt increments at 1 min each until the subject was unable to continue. After maximum effort the subject continued the no-load pedaling for 15 min. Oxygen (measurement by fuel cell) and carbon dioxide (measurement by infra red) contents of the expired air were determined at 15 sec intervals, as was heart rate.

An indwelling heparinized catheter was inserted in a forearm vein to facilitate blood sampling. Blood samples were taken before the test, just

TABLE 2												
	Max	Мах	Max		Laet	ate			Blood ;	glucose		
Test	Watts	V()2	HR	М	+5	+15	$_{ m pH}$	()	M	+ 5	+15	
1	300	-1.1	155									
2	330	4.2	162	-5	16	11	7.17	105	88	125	114	
3	360	4.1	159	4	15		7.16					
-1	390	4.4	160	8	16	13	7.19	103	87	115	108	
5	330	4.5	152	-5	14	9	7.26	93	74	94	78	
6	360	4.0	153	-5	12	12	7.22	85	73	80	94	(Tenor
												min)
7	320	4.5	138	7	12	_	7.26	127	102	97		(Tran

Max VO2 in 1/min; Max HR in b/min; Lactate in mmole/1 taken at Max, Max \pm 5 min, Max \pm 15 min; pH is the minimum value measured for blood pH; Blood glucose values in mmoles/dl taken before exercise, at Max, at Max \pm 5 min, and at Max \pm 15 min.

before the no-load pedaling, every 30 sec starting at the lactic acid threshold and until 5 min post-maximum effort, and a final sample 15 min postmax. Each 3 ml sample of blood was packed in ice and analysed within 4 hr. for pH, lactic acid, glucose, and potassium. An Instrumentation Laboratory Model 813 Blood Gas Analyser was used to measure the pH of the whole blood. Lactate, potassium, and glucose were measured on plasma, lactate with a DuPont Automatic Clinical Analyser and glucose and potassium with a Beckman Instruments ASTRA 8. Significant results of the stress tests and blood analyses are given in Table 2.

During stress tests #3 (no beta-blockade) and #6 (beta-blockade) impedance cardiography measurements were taken [43,44]; the calculated cardiac data for these two tests are given in *Table 3*.

From the oxygen consumption and carbon dioxide production data during recovery following maximum effort, the alactic oxygen debt, the rate of alactic oxygen debt recovery, and the rate of lactate recovery were calculated [45,46]. Results are given in *Table 3*.

Results and Discussion

The differences between tests #6 and #7 (beta-blockade) and the other tests (no blockade) are, except as

noted, in the directions that would be predicted. The subject was able to continue his normal running, albeit somewhat slower, during Tenormin treatment, but could not train during Trandate treatment.

datel

Heart rate: The HR on Tenormin treatment was reduced at rest and at all except the very highest work load. The difference was 15-20 beats/min up to intermediate loads. It was not expected that the difference would become less as maximum exercise was approached. The heart rate increased 18 beats/min during the last minute of test #6, but an average of only 6 beats/min during the final minute of the previous tests. HR recovery was also more rapid from test #6 than from the other tests.

During Trandate treatment no lowering of the resting HR was noticed. During test #7 the lowering of the HR was much less, but the previous maximum HR could not be reached.

Stroke volume: During test #4 the SV increased until about 70% of maximum work was reached, then declined. During test #6 the SV appeared to be increasing during the entire test, reaching a higher value at maximum effort than for #4. In fact the normal relationship between inotropic and chronotropic effects seems to be reversed under beta-blockade. During blockade the first response seems to be inotropic at all but max-

imum loads. This should not present any problem for any individual who has an adequate stroke volume reserve. This subject did comment that he was very aware of the heavy, slow beat of his heart while on Tenormin treatment but not while on Trandate treatment

Ventilation: The ventilation vs. oxygen consumption rate curves were not different from the other tests.

Oxygen consumption: During tests #6 and #7 oxygen consumption was as much as 25% lower at all but the highest work load, but increased rapidly to reach the same level as the other tests. In other studies [22,28] the subjects had not been able to reach their nonblockade levels of work or of oxygen consumption.

Blood glucose: Glu levels were lower for test #6 than for any other test at all work levels, and did not show the quick recovery after exercise. During test #7 the Glu level started higher than normal and declined steadily. It was not expected that hypoglycemia would become serious in only 15 min of exercise.

Blood lactate: LA levels during beta-blockade were the same at rest, but were slightly lower at all work levels than for the other tests. Calculated removal of LA as shown in *Table 3* was quite different during both beta-blockade tests.

Blood potassium: The normal quick increase in blood potassium during maximum exercise was observed during test #7 but not during test #6.

Quick energy use: We have found for normal, healthy subjects that the lactate recovery rate seems to be a good index of the level of conditioning, with higher values for those subjects who have had endurance training. These individuals also have high values for maximum work and for maximum oxygen consumption. We have also found that strength-trained individuals have high values of the alactate debt and alactate recovery rate.

The alactate (creatine phosphate)

debts were not significantly different during tests #6 and #7, nor were the alactate recovery rates, while the lactate recovery rates were distinctly higher. During both beta-blocker treatments the subject found no difficulty in performing quick maximum-effort exercise, such as weight lifting. Normal training for distance running was hampered during Tenormin treatment and was not possible during Trandate treatment.

Respiratory quotient: Tests #6 and #7 had the higher RQ over most of the test range, which is one indication of less free fatty acid metabolism.

Thus we have found two sources of discouragement of sustained physical activity for this subject. The lowered heart rate makes it more difficult but not impossible for the subject to reach an increased cardiac output. This was more pronounced during the Tenormin treatment. Even more important for this subject is the lessened availability of glycogen and free fatty acids to sustain activity after the first few minutes, and this was more pronounced during the Trandate treatment. It is to be expected that individuals who are less well conditioned than this subject will have even more difficulties with an exercise program while under treatment with beta-adrenergic blocking agents.

It might be noted that there are some similarities between the symptoms found for the metabolic myopathy described by McArdle [47,47] and the alterations in carbohydrate metabolism caused by the beta-blockers. These include normal muscle strength for short-term effort with the inability to continue long-term effort; in both cases the metabolic defect seems to be the inadequate utilization of muscle glycogen during exercise.

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				TABI	Æ 3							
	At 210 Watts											
Test	Alac Debt	Alac Recov	Lac Recov	Cardiac Output		End Diast Vol	Stroke Vol	HR				
1	2.6	2.0	0.17									
1 2 3	3.6	2.9	0.24									
3	2.7	2.31	0.3	18	70	200	140	132				
4 5	3.5	3.3	0.19									
5	3.5	2.6	0.25									
6	3.0	2.0	0.33	14	68	164	112	123	(Tenor- min)			
7	4.2	3.2	0.70						(Tran- date)			
*	1.5 - 2.5(.3)	1.0 - 2.0(.2)	0.1 - .4(.05)									

	TABL	E 3A			
		At	Max Wa	itts	
	Cardiae	Figet	End	Stroko	
				Vol	$_{ m HR}$
3	21	76	175	133	159
6	22	74	201	149	153

Alactic debt in 1 of 02, alactic recovery rate in 1 of 02/min, lactic recovery rate in 1 of 02/min. Cardic output in 1/min of blood, ejection fraction in percent, end diastolic volume in m1 of blood, stroke volume in 1 of blood, and heart rate in beats/min.

- * Average ranges (with standard deviations) for young man, 75 90 kg body weight; healthy, but not highly trained.
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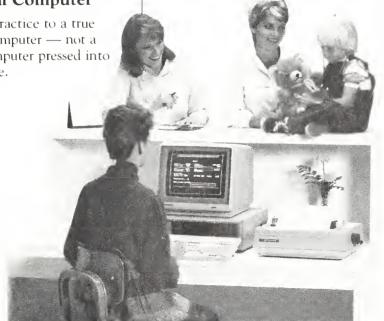
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Popliteal Artery Aneurysm: Case Report

RADE PEJIC, M.D. Michigan City

Abstract

Most common types of peripheral aneurysms are those involving the popliteal artery. Hypertension and arteriosclerosis obliterans are present in more than 50% of such patients. Also, a large percentage of such aneurysms are bilateral. Definitive diagnosis is made by arteriogor digital subtraction raphy angiography (DSA) if a pulsatile mass is felt on examination of the popliteal space. Appropriate surgical therapy is direct excision of the aneurysm with reversed saphenous vein graft interposition. If autogenous graft is not available, then a PTFE graft is acceptable for use. Postoperative recovery and long-term prognosis is excellent.

NEURYSMS OF THE popliteal artery are by far the most common of the peripheral type of aneurysms. This is so because the popliteal artery has less protection by muscles and is subject to frequent flexion and extension because of the adjacent knee joint. Consequently, frequent knee flexion combined with an arteriosclerotic popliteal artery can eventually produce dilatation of the vessel. Another pathogenesis is post stenotic dilatation, in view of the fact that the artery is compressed by the tendinous hiatus of the adductor magnus in the distal thigh and also at the level of the arcuate popliteal ligament behind the knee joint.

Three types of popliteal artery aneurysms are:

- Proximal: usually large and multilobular, occupying the space behind the femoral condyles.
- Middle: usually extend proximally and distally around the knee joint.
- Distal: smaller than the preceding two forms and usually silent until they thrombose.

Arterial hypertension is present in almost 50% of these patients and preexistent arterial occlusive disease in 60% of the involved limbs. The nature of the occlusion is usually arteriosclerosis obliterans, but in some cases the thromboembolism is the cause as a complication of the aneurysm. A large percentage of such aneurysms is also bilateral. The four basic surgical techniques in management of popliteal artery aneurysms are:

- Complete excision with reverse saphenous vein graft interposition, using end to end anastomosis.
- Partial excision and preservation of the collateral flow by intra-saccular



FIGURE 1: Lateral arteriogram of popliteal artery aneurysm. Full extent of the aneurysm is not appreciated because of intraluminal thrombus.

suturing of the ostia and graft interposition.

- Exclusion of the lesion using a vein graft bypass with proximal and distal ligation of the aneurysm in order to avoid thromboembolic complications to the distal arteries.
- Lumbar sympathectomy combined with aneurysmectomy and vein graft interposition to provide better collateral flow, especially in the presence of distal occlusive disease.

The technique of ligating the aneurysm proximally and distally and bypassing it by a conduit has been advocated by Edwards. However, progressive expansion of the remaining aneurysm has been reported, which necessitates additional surgery. The author prefers complete excision of the popliteal artery aneurysm with vein graft interposition.

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This surgical approach is the most definitive, eliminating all pressure sensations upon the tibial nerve, the knee joint, and the popliteal veins. Consequently, there is no need for any additional surgical procedures and the graft failure is minimized by using autogenous saphenous vein, rather than Dacron or PTFE prosthesis.^{7,8}

Case Report

A 79-year-old white man presented with a pulsating mass in the right popliteal area of several months duration. The patient complained about a pressure sensation behind the right knee and weakness of his right leg upon ambulation. There was no history of claudication and the mass was painless.

PMH: Has been hypertensive for 10 years; there is no history of diabetes, angina or myocardial infarction. The patient is a two pack a day smoker (for 40 years). PE: BP: 160/92; P 84; R 20; T 98.6°. The physical findings were negative except for those localized in the right lower extremity. A right femoral bruit was heard over the common femoral artery and the patient had a pulsating

elliptical mass behind the right knee which measured approximately 8×5 cm. Bilateral femoral pulses were +1, the popliteal +2, and the pedal pulses +1. An arteriogram confirmed the presence of a large popliteal aneurysm with three vessel run-off.

At surgery, an hour-glass shaped aneurysm was completely excised via the posterior approach and a vein graft interposition was performed from the superficial femoral artery at the adductor canal to the distal popliteal artery. Postoperatively, the patient did well except for a cellulitis around his surgical incision and was discharged on his 14th postoperative day. He was able to ambulate without any difficulty and had the same pedal pulses postoperatively.

Discussion

Whenever a popliteal mass is found on physical examination, the differential diagnosis should include a lipoma, fibroma, synovial cyst, enlarged sebaceous cyst and also a popliteal artery aneurysm. One should always listen for a bruit over the mass and examine the peripheral pulses bilaterally. Conventional arteriography or



FIGURE 2: AP view of popliteal artery aneurysm showing its tortuous course. Intraluminal thrombus is also present.



FIGURE 3: Excised popliteal artery aneurysm. External diameter is larger than that seen in the arteriogram because of intraluminal thrombus material.

digital subtraction angiography (DSA) may be used to definitively establish the diagnosis. If there is still doubt because of intra-arterial thrombus formation, then an ultrasound of the popliteal artery could be performed to measure its cross sectional diameter.

The basic predisposing lesion for such an aneurysm is arteriosclerosis. This process weakens the arterial wall, which is normally elastic, and accelerates premature degeneration of the arterial intima and media. Other contributing factors are hypertension, smoking and diabetes. Thus, when an arteriosclerotic artery is subjected to chronic flexion and extension, it may weaken even more and produce an aneurysm.

The preferred treatment of choice is complete excision of the aneurysm

with reversed saphenous vein graft interposition. Proximal and distal ligation of the aneurysm with partial resection and graft bypass is an acceptable alternative in those cases where complete excision is deemed hazardous because of tibial nerve or popliteal vein adherence to the aneurysm sac secondary to intense cicatrix formation.

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PUBLIC HEALTH NOTES

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risk group have higher geometric mean serum PCB levels than the unexposed group or those previously studied general populations.

As measured by the proportion of high risk group subjects having serum PCB levels greater than or equal to 20 parts per billion, it appears that exposures to this group from nonoccupationally related sources were no different from those experienced by those considered to be at lower risk or from a representative sample of at-risk persons around these three waste sites in Bloomington. However, the study suggests that the geographic area related to these three contaminated sites may represent one which is characterized by generally higher average human serum PCB levels compared to that expected based on previously characterized populations in other areas of the United States.

Because of statistical constraints, the ISBH could not implicate any specific environmental pathway from these three waste sites as being the exposure routes of most significance, except documented occupational exposures and, possibly, from scavenging of capacitor parts.

Overall, the ISBH found no excesses of self-reported, physician-di-

agnosed adverse health effects in nine key target organ systems which included skin, liver, and nervous. However, there was a statistically significant dose-response relationship between serum PCB levels and the occurrence of self-reported high blood pressure; this relationship remained statistically significant when data were controlled for possible confounding effects of age or smoking. The association with high blood pressure has been reported previously in a population that was exposed to PCBs through ingestion of contaminated fish. Also, there were isolated statistically significant findings from the clinical chemistry tests when compared to the serum PCB measurements, some of which may be consistent with liver disease but were not corroborated in further analyses.

Conclusions should not be drawn until all of these findings can be further evaluated in larger epidemiologic studies, specifically designed to elucidate the relationships of PCBs to these symptoms or organ systems.

In summary, the most striking finding of this pilot investigation is that the population in this geographic area appears to have higher average human serum PCB levels compared to previously characterized populations

in other areas of the United States.

Exposures in this community require further evaluation—perhaps in a comparison of a representative population-based sample from Bloomington, Indiana, to a truly nationally representative population sample. A key area to be elucidated is the specific environmental pathway or combinations of pathways and mechanisms via which exposures have occurred to these three sites, as well as to other possible local sources.

Finally, although the range of serum levels reported herein from exposures to PCBs in the general environment are lower than those which have been associated with acute symptoms or illness in other studies. it is not known whether these levels are associated with long-term health risks. Associations of such chronic, low-dose exposures with observable health effects must be evaluated further before any conclusions can be drawn. Because of this uncertainty, there is a continuing need for appropriate remedial action to prevent further exposure to these populations.

For more information about the pilot study, contact the Chronic Communicable Disease Control Division, Indiana State Board of Health—(317) 633-8554.

Synopses of NIH Consensus Reports

Lowering Blood Cholesterol to Prevent Heart Disease

The National Institutes of Health recently published a brochure which summarizes all of the literature to date regarding the relationship between high cholesterol levels and coronary artery disease.

There are approximately 550,000 deaths in the United States annually which are directly related to coronary artery disease. A number of risk factors have been identified, including cigarette smoking, high blood pressure, male sex, obesity, diabetes mellitus, physical inactivity, high cholesterol levels, and even both genetic and personality factors.

The authors feel that there is unquestionable evidence that high cholesterol levels are a significant factor in developing coronary artery disease. The high density lipoprotein (HDL) seems to be protective while low density lipoprotein (LDL) seems to be the main offending factor. They concluded that we should attempt to lower cholesterol levels that are 240 mg. per cent or above in patients age 40 or over, and that we should treat patients in their 20's if they have a very strong family history and their cholesterol levels are above 200 mg. per cent.

The first method of treatment, of course, is a dietary regimen and trying to lower the weight of those who are obese. Of course, we all recognize the

fact that this is most often unsuccessful because of the patient's refusal to adhere to prescribed diets. In the future, there will be more emphasis placed on trying to work with the entire family as well as working with restaurants, especially the fast food chains and school cafeterias, to try to provide lower fat diets. There is on-going research studying cholesterol at the cellular level.

This is a very interesting brochure and could be recommended for all physicians in clinical practice.—I.E. Michael, M.D., Indianapolis

Fresh Frozen Plasma: Indications and Risks

Use of plasma and its products have developed over four decades.

The use of FFP has increased 10 times within the past 10 years and has reached two million units per year.

Although well defined, indications exist for use of FFP in single or multiple coagulation deficiencies; indications for many of its uses are empirical.

In order to resolve some of the questions as to the rising use of FFP, a consensus conference was held by the National Institutes of Health, the FDA, and the Office of Medical Applications of Research.

The following questions were posed:

- 1. What are the current recommended clinical indications for FFP?
 - 2. What are the risks for FFP?
- 3. What alternative therapies ex-
- 4. What is the current knowledge as to the effectiveness of FFP?
- 5. What directions for future research are indicated?

The conclusions were that the administration of FFP has increased

markedly in recent years despite lack of evidence for its use in most circumstances. This increase has occurred despite the increased evidence of potential risks, including viral hepatitis and AIDS. Use of FFP must be justified on clinical grounds until further data is available. Research to develop safer FFP and other alternatives is stressed. It was specifically stated that there is no justification for the use of FFP as a volume expander or as a nutritional source—safer alternatives are available.

FFP is indicated for specific coagulation protein deficiencies, selected patients needing massive transfusions, patients with multiple coagulation defects (as with liver disease) in association with plasma exchange in TTP, for infants with protein losing enteropathy, and in patients with selected immune deficiency states.—

John A. Cavins, M.D., Indianapolis

Treatment of Hypertriglyceridemia

A conference was recently held by the National Institutes of Health on the treatment of hypertriglyceridemia and the following is a summary of the findings.

Triglyceride levels below 250 mg./dl in presence of a normal cholesterol probably do not increase the risk of cardiovascular disease, and generally respond to exercise and diet. Fasting plasma triglyceride levels between 250 and 500 mg./dl may be normal or serve as a marker for increased risk. In other words, elevated triglycerides in this range may not raise the possibility of other disease states such as diabetes mellitus, chronic renal

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These synopses are based on NIH consensus development conferences conducted recently. Complete consensus statements are available from the Office of Medical Applications of Research, National Institutes of Health, Bldg. 1, Room 216, Bethesda, Md. 20205.

Blueprint for Creating an 'Open Assembly' ISMA Hospital Medical Staff Section

HE ISMA HOUSE OF DELEGATES, during its October meeting in 1984, took a giant step forward on behalf of Hoosier physicians by adopting a resolution calling for the formation of a Hospital Medical Staff Section for the Indiana State Medical Association. In so doing, the House of Delegates recognized the needs of all physicians who relate to hospitals through their daily practice patterns and by the fact that they are a member of at least one hospital medical staff. The needs of Indiana physicians who serve on various hospital medical staffs become more complex due to the changing medical care environment affecting our hospitals. These changes have caused us to further examine the trends that will shape hospital structure and management in the 1980s and to become more aware of the issues and problems that are unique to our hospitals and our 6,000 physician members that serve them.

As I monitored the discussion that occurred in our reference committee, dealing with the subject of a Hospital Medical Staff Section for ISMA, I shared the conviction that there is a need to provide the counsel and services necessary to strengthen the local hospital medical staff in each of our communities. The past decade has witnessed a continuing increase in multi hospital systems, as well as a substantial growth in the number of "for-profit" hospitals. This is all occurring at a time when there is already significant dependence on the hospital and its technologies for the delivery of medical care. AMA sur vey studies indicate an ever-increasing number of physicians entering into full time, part time, or negotiated



LAWRENCE E. ALLEN, M.D.
President
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contractual relationships with hospitals and other health care facilities. The results of the AMA survey study conducted between October and December of 1981 indicated that approximately 191,000 physicians had contracts with hospitals or other health care facilities. The trends in changing hospital structure and management observed thus far in the 1980s would indicate that multiple hospital systems are expected to expand rapidly throughout the '80s and by the end of the decade it is projected that 50% to 60% of all hospitals may be part of some multi-unit system. It is also expected that hospitals attempting to increase revenues will seek to expand services, particularly in the ambulatory care

area, such as surgi-centers and satellite clinics. Hospitals are already seen as an ever-expanding competitor for physicians in private practice. All of these changes are bringing a new dimension to medical staff hospital relationships.

Physician profiles indicate that 56% of all office-based physicians spend over 20% of their practice hours in hospitals. Since hospitals are where physicians spend a significant portion of their practice time, hospitals must also present the local arena where physicians individually and collectively may discuss and resolve their problems resulting from changes that affect hospitals and physicians alike. It is easy to understand why physicians are apprehensive in situations where the medical staff may not have sufficient voice in hospital planning or in hospital governing board decisions, which may directly affect changes in hospital ownership, competition between hospital ambulatory services, and office-based physician practices, hospital diversification into other profit-making activities, closed medical staff arrangements and/or exclusive contracts, interpretation and compliance with JCAH standards, jurisdiction in delineation of privileges and hospital cost containment activities, to name a few.

The medical care environment is changing in ways that will increase the need for representation of medical staffs in the formation, development and implementation of new policies in the area of physician-hospital relationships. It should be clearly obvious to all that the role of organized medicine, in view of such environment of change, resides with the

responsibility to innovate and co-ordinate such activities as the formation of a Hospital Medical Staff Section.

The officers and staff of ISMA have initiated the organizational efforts leading to the creation of a Hospital Medical Staff Section for ISMA. It is our wish and the intent of the Board of Trustees to develop a representative structure which will provide a system (1) to solve problems and avoid polarization of medical staffs, (2) to identify the implications of future trends on the role of physicians individually and as members of medical staffs, (3) to acquaint medical staff leadership with policy in organized medicine, (4) to develop information on issues of common concern to medical staffs, and (5) to distribute such appropriate information to each hospital staff unit, and more or less provide an easy contact point for medical staff leaders with ISMA resources. It is hoped that such a structure of approach to communication will facilitate bi-directional communication and that the ISMA-HMSS will provide a forum for medical staff representatives to meet annually and discuss common issues with the ISMA House of Delegates.

It is the contention of the Indiana State Medical Association that the degree of physician involvement with hospitals must continue to increase. Physicians can no longer be concerned solely with issues that affect their individual practices and must involve themselves with the collective concerns of the hospital medical staff and thus seek out the most effective channels to deal with these concerns. The hospital medical staff has a unique opportunity to make contributions to the continued development of quality patient care in the institutional setting. It is to this end that the ISMA Hospital Medical Staff Section will direct its energies so as to lend strong and effective support to individual hospitals as county medical societies and your State Medical Association work together to provide the counsel and services necessary to strengthen the local hospital medical staff. I commend the House of Delegates and encourage the establishment of an effective Hospital Medical Staff Section for the Indiana State Medical Association.

Agenda for Creation of an ISMA Hospital Staff Section

During the initial development of the Hospital Medical Staff Section, flexibility is desirable and necessary. The following rules and suggestions are stated at this time and can be incorporated in the subsequently enacted bylaws for the Hospital Medical Staff Section:

RULE 1: Representatives shall be members of the ISMA.

RULE 2: Initially, representatives to the Section may be selected by the Executive Committee of the medical staff; however, after January of 1986, proof of nomination and election by members of the active voting medical staff shall be required.

RULE 3: Representatives shall be active voting members of the medical staff with clinical privileges at the hospital.

The following suggestions are made at this time to facilitate the formation of the Hospital Medical Staff Section, and may or may not be a part of our subsequent bylaw structure for the section:

SUGGESTION 1: Representative should be someone who could require experience and provide continuity of representation for a period of two or more years.

SUGGESTION 2: The representative should be someone who can commit the time and effort to this important task and fully participate. Because of the heavy time and meeting commitments, strong consideration should be given to someone other than the current ISMA delegate or alternate delegate.

SUGGESTION 3: To facilitate bidirectional communication, the representative to the section ideally should become a member of the Executive Committee of the medical staff, perhaps by appointment.

SUGGESTION 4: A mechanism should be established whereby the representative of the hospital medical staff regularly communicates with the hospital medical staff and the governing body of the hospital.

SUGGESTION 5: The representative should communicate on a regular basis with the local county medical society.

Model for the Hospital Medical Staff Section Assembly

The open assembly model is considered the best potential forum for representatives to the hospital medical staff section; nevertheless, opportunity for further modification of the open assembly model to accommodate the governing council and general representation to the Hospital Medical Staff Section can be defined through subsequent modification of bylaw structure in compliance with the ISMA constitution and bylaws.

Financing for the expenses of the Hospital Medical Staff Section governing council will be borne by the Board of Trustees of ISMA through the budget of the ISMA House of Delegates. The expenses of representatives to the ISMA-HMSS assembly should be financed through sources made available at the local hospital level.

The Hospital Medical Staff Section assembly should be attended by one representative or his alternate.

The following is an agenda for creating the governing council of the ISMA Hospital Medical Staff Section and providing for hospital appointment of representatives and their alternates to the section:

(1) An organizing committee made up of representatives from Indiana to the AMA Hospital Medical Staff Section will meet and form a nominating committee for the governing council. The governing council will direct programs and activities of the section, subject to the approval of the ISMA Board of Trustees. Members of the governing council will consist of the officers, section delegate and alternate delegate to the ISMA House of Delegates (section delegates are nonvoting members of the ISMA House of Delegates), and two at large governing council members elected at the business meeting of the section. The officers of the section shall have the following duties and responsibilities:

CHAIRMAN: Shall preside at the business meetings of the section and at the meetings of the governing council.

VICE-CHAIRMAN: Shall assist the chairman and preside in the absence of the chairman, or at his request.

SECRETARY: Shall maintain such records as may be necessary to, or advisable for the conduct of, the activities of the section.

DELEGATE AND ALTERNATE DELEGATE: Shall represent the members of the section in the ISMA House of Delegates as non-voting members. The term of service for governing council members, including the delegate and alternate delegate, shall be that of two years, beginning at the conclusion of the annual meeting at which they are elected, and ending at the conclusion of the next annual meeting. It may also be advisable to adopt a provision for staggered terms and to allow for reelection to unlimited successive terms.

The organizing committee will present a slate of nominees for each

office at the first business meeting of the ISMA Hospital Medical Staff Section, to be conducted in Indianapolis in October of this year. Further nominations for these offices will be appropriately received from the floor of the assembly. The organizing committee and its chairman will conduct the proceedings having to do with the nominating process until the chairman of the governing council is elected and can assume the duties of his office.

Within the staff capability of ISMA, I have enlisted the promotional services of Beckett Shady-King, to whom future correspondence may be forwarded. I am further gratified with the willingness of Dr. Franklin Bryan to assist us in the formation of the ISMA-HMSS.

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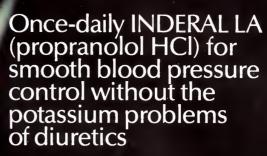
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Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

Once-daily beta-1/beta-2 INDERAL LA blockade (PROPRANOLOL HCI) LONG ACTING CAPSULES



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80 mg 120 mg 160 mg

Please see brief summary of prescribing information on the next page for further details.

Once-daily For beta-1/beta-2 NDERAL LA

(PROPRANOLOL HCI) LONG ACTING CAPSULES

RRIFF SUMMARY (FOR FULL PRESCRIBING INFORMATION. SEE PACE AGE CIRCULAR.)

INDERAL* LA brand of propramolol hydrochloride (Long Acting Capsules)

DESCRIPTION. Inderat LA is formulated to provide a sustaining release of propramolol hydroschloride release of propramolol hydroschloride. Inderat LA is available as 80 mg. 120 mg. and 160 mg. capsules.

CLINICAL PHARMACOLOGY, INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor situaliting agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL the chronotropic, inotropic and vasodiator responses to beta-adrenergic stimulation are decreased proportionately. INDERAL LA Capsules (80. 120. and 160 mg) release propramolol HCI at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propramo of plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets. The lower AUCs for the capsules are due to greater hepatic metalosism of propranolol resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

propriation l'estaining florimine silvent race traduscription of piopination (12) hours then decline exponentially INDERAL LA should not be considered a simple mg for mg substitute for conventional proprianolo and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional proprianolo is a possible need for retitration upwards should be considered especially in maintain effectiveness at the end of the dosing interval. In most clinical settings however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect. INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL LA as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period. The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output. (2) inhibition of remin release by the kidneys and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase influshly in readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum potassium concentration when used in the teatment of hypertensive patients.

In angina pectoris propranolol generally reduces the oxygen requirement of the heart at an unusuella of effect by blockage, the category and the resistance of the heart at a control patient of the period propranolol generally reduces the oxygen requirement of the heart at

been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive palients. In angina pectoris propranolol generally reduces the oxigen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate systolic blood pressure and the velocity and extent of myocardial contraction. Propranolol may increase oxigen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period. The net physiologic effect of beta-adrenergic blockade is usually, advantageous and is manifested during exercise by delayed onset of pain and increased work capacity. In dosages greater than required for beta blockade. INDERAL also exerts a quinidine-like or anesthetic-like membrane action in the treatment of arrhythmias is uncertain. The mechanism of the antimigraine effect of propraholol has not been established. Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain. Beta receptor blockade can be useful in conditions in which because of pathologic or functional changes, sympathetic activity is detrimental to the patient. But there are also situations in which sympathetic stimulation is viral. For example, in patients with severely damaged hearts adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved. In the presence of AV block, greater than tirst degree, beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients with adrenergic bronchodilator activity which should be preserved in patients with supplication to proprianolos, so its significantly dialyzable.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the manage ment of hypertension it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the living-term management of patients with angina pectrus.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of proprianolol in the freatment of a migraine attack that has started has not been established and proprainolol is not indicated for such use. Hypertrophic Subaortic Stenosis: INDERAL LA is iseful in the management of hypertrophic subaortic stenois: especially for treatment of exertional or other stress-induced angina palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of proprianol oil hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is confraindicated in 1) cardiogenic shock. 2) sinus bradycardia and greater than first degree block. 3) bronchial asthma. 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a fachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component porting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure if necessary they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitals and diurelics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart secretary.

muscle
IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE continued use of beta blockers
can in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart
failure, the patient should be digitalized and/or treated with diuretics, and the response
observed closely, or INDERAL should be discontinued (gradually, if possible).

IN PATIENTS WITH ANGINA PECTORIS there have been reports of exacerbation of angina and, in some cases myocardial infarction following abrupt discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be rautioned against interruption or cessation of therapy without the physician's advice if INDERAL therapy is interrupted and exacerbation of angina occurs it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized it may be prudent to low the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranoiol for other indications.

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema) PATIENTS WITH BRONGHOSPASTIC DISEASES SHOULD IN GENERAL NOT BLOCK ERS. INDERAL should be administered with caution since it may blo

tion profued by endogenous and exogenous catecholamine stimulation of beta receptors

MATOR SURGERY. The necessity or desirability of withdrawal of beta-blocking therapy
prior to major surgery is controversial. It should be noted in however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthe sia and original procedures



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INDERAL (propranofol HCI) like other beta blockers is a competitive inhibitor of beta receptor agonists and its effects can be reversed by administration of such agents, e.g. dobular-mel or isoproterenol. However such patients may be subject to protracted severy hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with

a blockers
DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the ap

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) cacute hypoglycemia in abile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin. THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptom of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests. IN PATIENTS WITH WOLEF-PARKINSON-WHITE SYNDROME, several cases have bee reported in which, after propranolol, the tachycardia was replaced by a severe bradycardi requiring a demand pacemaker. In one case, this resulted after an initial dose of 5 micropranolol.

PRECAUTIONS. General Propranolol should be used with caution in patients with impaire hepatic or renal function. INDERAL (propranolol HCI) is not indicated for the treatment of hypertensive emergencies.

hypertensive emergencies Beta adrenoreceptor blockade can cause reduction of intraocular pressure. Patient should be fold that INDERAL may interfere with the glaucoma screening test. Withdrawal malead to a return of increased intraocular pressure. Clinical Laboratory Tests. Elevated blood ureal levels in patients with severe heart disease elevated serum transaminase, alkaline phosphatase, actate dehydrogenase. DRUG INTERACTIONS. Patients receiving catecholamine-depleting drugs such as rese pine, should be closely observed if INDERAL is administered. The added catecholamine blocking action may produce an excessive reduction of resting sympathetic nervous activit which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility. Long-term studies in animals hav been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosag levels. Reproductive studies in animals did not show any impairment of fertility that was

levels. Reproductive studies in animals did not show any impairment of leftility that wa attributable to the drug. Pregnancy. Pregnancy Category C. INDERAL has been shown to be embryotoxic i animal studies at doses about 10 times greater than the maximum recommended human dost. There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetur. Nursing Mothers. INDERAL is excreted in human milk. Caution should be exercised whe INDERAL is administered to a nursing woman. Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and having represented to the properties of the properties. Provided the withdrawal of therapy. Cardiovascular bradycardia congestive heart failure intensification of AV block hypotension paresthesia of hands thrombocytopenic purpura arterial insufficiency, usually of the Raynaud type.

Raynaud type

Central Nervous System lightheadedness, mental depression manifested by insomnic lassifude weakness fatigue reversible mental depression progressing to catatonia, visu-disturbances, halifucinations an acute reversible syndrome characterized by disorientation fit time and place short-term memory loss emotional lability slightly clouded sensorium an decreased performance on neuropsychometrics.

Gastrointestinal nausea vomiting epigastric distress abdominal cramping diarrheiconstipation mesenteric arterial thrombosis, ischemic colitis.

Allergic pharyngitis and agranulocytosis erythematous rash fever combined with achin and sore throat laryngospasm and respiratory distress.

Respiratory bronchospasm

Hematologic agranulocytosis northrombocytopenic purpura, thrombocytopen

Hematologic agranulocytosis nonthrombocytopenic purpura, thrombocytopen

Auto-Immune In extremely rare instances systemic lupus erythematosus has bee

reported Milicellaneous allopedia LE-like reactions psoriasiform rashes dry eyes male implience and Peyronie's disease have been reported rarely. Oculomucocutaneous reaction involving the skin, serous membranes and conjunctivae reported for a beta blocker (practoic have not been associated with propranoiol.)

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride i

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in sustained-release rapsule for administration once daily if patients are switched from INDERA tables to INDERAL LA capsules care should be taken to assure that the desired therapeut effect is maintained. INDERAL LA should not be considered a simple mg for mg substitute in INDERAL INDERAL LA has different kinetics and produces lower blood levels. Retitration mabe necessary especially to maintain effectiveness at the end of the 24-hour dosing intervences are recommended in the produced of the pro

(see WARNINGS) MIGRAINE — Dosage must be individualized. The initial oral dose is 80 mg INDERAL to once daily. The usual effective dose range is 160-240 mg once daily. The dosage may to increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response sit no bitained within four to six weeks after reaching the maximum dose. INDERAL LA theral should be discontinued. It may be advisable to withdraw the drug gradually over a period count to be a second without the drug gradually over a period count to the second without the drug gradually over a period count to the second without the drug gradually over a period count to the second without the second

several weeks
HYPERTROPHIC SUBAORTIC STENOSIS 80-160 mg INDERAL LA once daily
PEDIATRIC DOSAGE At this time the data on the use of the drug in this age group are to
limited to permit adequate directions for use

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As He Grew
Older, the Author's
Golf Skills
Declined, So He
Invented a Way
to Cope with
Those Otherwise
Depressingly High
Scores . . .

became older my skill declined. I rapidly went from poor to bad, from bad to terrible and from terrible to really awful. I considered my stance to be excellent, my swing really quite good and my follow-through as perfect as my aging body allowed. But my score showed none of these facts. My score card was double-digit inflation right down the line. It was discouraging. It was worse than discouraging; it was depressing. I truly loved my sport, but I hated my score and what it told about me.

One night I sat at home trying to decide if I should give up the game which had so obviously given up on me. Suddenly I had a revelation. The answer, when it came, was so reasonable, so clear, so very right, that I wondered why no one had thought of it before. It was simple. So very simple! Keep the game ... CHANGE THE SCORING!

An hour or two spent with paper and pencil resulted in a whole new life for me as a golfer. No longer am I ashamed to show my score eard. My score now stands out proudly against the card. I do not cheat. My system does not use numbers. It uses letters. Letters of the alphabet. And only the first three letters of the alphabet at that. Here is how it works:

If you complete a hole of golf and do not lose any clubs you cannot get lower than a C on that hole. How well can you score? Theoretically, you can get an A+. To do that you would have to shoot a bogey. Possible, of course, but not at all likely. (The possibility of shooting par or birdie is so far-fetched that it would be unthink-

able to include it in this paper on "Scoring for Senior Citizens." So there are not any A+'s. However, any other grade is possible.

Example: Say the first hole is a par 4 according to the old system of scoring. You, of course, take an 8. Let us say that one of those 8 strokes was a real beauty. The 5th stroke ... the one that reached the green. It looked good. It felt good. It was good! A real professional shot! You just know that when you get home and recall the 1st hole you'll think only of that beautiful approach to the green. On that hole you're surely entitled to a B ... possibly even to a B+ if you found a ball. (I forgot to mention you can go up to the next higher score if you find a ball. Needless to say, there is no penalty for losing a ball ... or going out of bounds ... or taking a Mulligan, either.)

Another example: The 3rd hole is a par 3, again according to the old system. You take your usual 7. Your second drive (remember, no penalties for Mulligans!) was gorgeous. It almost cleared the water. You could reach it with your wedge from the side nearest the green, it came that close to clearing. So what if you did drive from the ladies' tee? It was one hell of a poke. You'll think back on that drive with pleasure for a long time. An easy B+ by itself, right?

One last example: The 9th hole is a par 4 and you end up with a 9. But wait! You're having a good round. You were on the green in 5. Now number 9 has a tricky green. A very tricky green. It's like those greens they have in Scotland ... big and wavy and sloped every which way.

Correspondence: 1619 E. Jefferson Blvd., South Bend, Ind. 46617.

On that impossible green you sink a 10-foot putt! An honest-to-God 10-foot putt! No matter that it was your 4th putt! On that miserable 9th green you actually sunk a 10-foot putt! Your longest putt in seven years. Now, be reasonable. When you think back on the 9th, what will you think of? The eight strokes that were nothing or that magnificent putt? Worth an A any day.

In the Sandock System of Scoring there is one last thing you should know about totaling your score. If, in adding your score, you discover it was one of the best rounds you've had in a long time you may award yourself an A even if no A appeared for any hole. Golf is a game of honor and pride. Honor yourself! Be proud! You're entitled!

Now when you show someone your score card it will look like the illus-

	YUS	370	375	216	474	220	355	406	505	285	3206
	PAR	4	4	3	5	3	4	4	5	4	36
	HOLE	1	2	3	4	5	6	7	8	9	TOTAL
J	00	4	3	4	6	5	4	5	6	4	41
40	uk	5	4	4	5	4	5	4	5	5	41
L	00	B+	B	B+	(+	B	\mathcal{B}_{t}	B	B	B+	A-
M	el	4	4	3	6	3	5	5	6	6	42

tration above.

Be honest. Doesn't your score look better now compared to your buddies' scores than it would if you had put down every stupid stroke?

One more thing. This system is based on 9 holes only because that is all I can play at one time. However,

this system can be used for 18 holes by merely raising your score one full grade for each hole on the back 9. Most players using this system are quite pleasantly surprised at how very well they play the back 9 at their age. You will be, too. Have a good one!



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EDITORIALS

Comments on Babesia microti Article

I am writing in regard to the article, "Bahesia microti in an Indiana Woman," by Smith and Clevenger appearing in the March 1985 issue.

As stated, normal individuals often have self limiting illnesses that can be treated symptomatically. While they suggest that pentamidine may be beneficial, the drug has been ineffective in animal chemotherapeutic trials.

Recent use of exchange transfusion has been successful in decreasing parasitemia and can be lifesaving in patients with severe infections. The most efficacious drug regimen appears to be clindamycin in combination with oral quinine. The regimen is effective in human and in animal models. Quinine by itself has no activity but appears to potentiate the effects of clindamycin when given orally.

A few points should be considered when evaluating a patient with suspected babesiosis. One-third of reported cases of babesiosis occur in asplenic hosts. The spleen appears to play an important role in clearing the parasitemia and the most severe cases have been reported in its absence. Transfusion babesiosis has also been reported. and freeze thawing does not eradicate the parasite. Interestingly, the transmission vector



fine-lle laughs at all my jokes."

Lxodes dammini, or hard bodied tick, is also the vector for transmission of Lyme disease. Simultaneous infection has been reported.

It appears that only severely symptomatic patients should be treated and clindamycin and quinine would be the most effective treatment at this time. Exchange transfusion is effective in promptly decreasing parasitemia.—Ken Cornetta, M.D., PGY III, Dept. of Medicine, Indiana University School of Medicine.

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Babesia microti: The Authors Reply

We greatly appreciated the interest of Dr. Cornetta in our article and his points are well taken. However, we do take exception to the comment that pentamidine is without effect in animal models, although the article referenced does show pyrimethamine to be without efficacy in Babesia infections. Regarding pentamidine the article states: "Aromatic amines, including diminazene aceturate and pentamidine isethionate, have some efficacy in treating experimental B. microti infections in rodents." Pentamidine has also been shown to decrease parasitemia in humans.

We do agree, at the present time, that in seriously ill patients, a combination of clindamyein and oral quinine should be the drugs of first selection.

Of additional interest in this disease is the recent report of a new focus of *Bahesia microli* found in Wisconsin. This article further produces

serologic evidence of a second case of concurrent Lyme disease and Babesia microti.

Finally, it is important to note that automated blood counters will not detect the intracellular inclusion of babesiosis. This demands that in suspected cases of babesiosis and fevers of unknown origins, blood slides be microscopically reviewed by a trained examiner.—Darryl R. Smith, M.D. and Robert R. Clevenger, BS MT (ASCP), Parkview Memorial Hospital, Fort Wayne

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On Poverty, Help, and Social Programs

Guest Editorial

We all recognize that poverty exists in our U.S.A. We taxpayers have contributed uncounted billions of dollars in the past, and do so on a continuing basis trying to eliminate it. Since we have achieved so little with so many dollars, the time is past due for further study of the problem, and of why our efforts have been such a failure.

Officially, poverty is defined as family income below a certain arbitrary amount. Such arbitrary figure could have been set at one-tenth or 10 times that arbitrary amount; it would still be no less arbitrary, and it would still not be a good definition. I am uncertain that poverty can be satisfactorily defined.

Poverty is not the same as being poor. Some of us will admit that we grew up in families that were poor, and would deny that we grew up in poverty. For some of us, being poor provided an incentive to work harder to improve our status; to help ourselves to better our lot.

Some of our poorest citizens are one-parent households with small children. Many of these are families where the father contributes exactly what tom cats and stray dogs contribute to the support of their offspring. Some of these one-parent households are headed by immature teen-age girls who sought pregnancy as a means of getting out of uncomfortable living situations and onto the welfare rolls - not realizing that such is often the route to the poorest-ofthe-poor-type of poverty. In these cases, our present welfare system has actually encouraged such status. There is slight chance that these people can ever escape their poverty because they have no knowledge of how to do so or incentive to do so.

When one looks for poverty, one may look into the slum areas of our large cities because that is where poverty seems to be concentrated. These slums were not *constructed* by

anyone; they were created by slum dwellers, and usually created out of good, solid, expensive housing. Where slums have been destroyed, usually new ones were created elsewhere in a short time by the slum dwellers. It seems so futile for society to try to eliminate the slums, when the slum dwellers are so adept at creating new ones.

In medicine, we recognize that illnesses and situations occur where we cannot achieve cures; in some cases we cannot even achieve very good palliation. We deal with such problems daily. It has been suggested that we should limit our use of medical resources in some of the hopeless cases. I see a comparable situation in our socio-economic system relationship with "the poor." Many are, and are likely to remain hopeless, incurable cases despite all our tax-paid dollars. So, to what extent should we, as a society, by taxation, drive our middle class into poverty in the attempt to eliminate poverty?-L. A. Arata, M.D., Shelbyville

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CME QUIZ.

TO OBTAIN ONE HOUR OF CATEGORY 1 AMA CME CREDIT, answer the following questions by circling the correct answer on the answer sheet below. Complete and clip the application form and mail it to: Indiana University School of Medicine, CME Division, Fesler Hall 224, 1120 South Dr., Indianapolis 16223.

Prolonged Apnea in Infancy

CONTINUED FROM PAGES 561-566

- 1. Normal breathing patterns in infants include all but one of the following:
 - a. Periodic apnea
 - Transient apnea associated with choking on mucus
 - A 10 second apnea pause as sociated with minimal eyanosis
 - A 15 second apnea pause fol lowing a sigh during sleep
- 2. A clinically significant color change includes all but one of the following:
 - Plethora associated with prolonged crying
 - Mild transient evanosis involv ing the entire face
 - Pallor associated with brady
 - d. Cyanosis and transient respi ratory distress after an epi sode of vomiting
- Acceptable components of the man agement of patient with apnea of infancy may include:
 - a. impedance pneumography

- b. theophylline
- caffeine
- all of the above
- 4. Family members caring for the child who is monitored in the home must
 - side effects of theophylline
 - cardiopulmonary resuscitation techniques
 - that apnea is the leading cause of SIDS
 - that the therapeutic range of theophylline is 8-12 mg/L
- 5. An acceptable starting dose of theo phylline in a 2 week old child is:
 - a. 4 mg/kg/day
 - b. 8 mg/kg/day
 - c. 12 mg/kg/day
 - d. 16 mg/kg/day
- 6. Infants who have prolonged apnea associated with a seizure disorder uniformly have:
 - a. an abnormal EEG
 - a need for theophylline in ad

- dition to anticonvulsive ther
- an abnormal polysomnogram
- apnea only during sleep
- none of the above
- 7. Gastroesophageal reflux in neonates and young infants:
 - is uniformly pathologic
 - is always associated with an abnormal contrast radiograph of the upper gastrointestinal tract
 - may be associated with ob structive apnea
 - never requires treatment as it is always benign and self limited
- 8. Criteria for discontinuing the home monitor include all but one of the following:
 - No apena alarm in the previous 2 months
 - A viral illness or immunization in the previous 2 months
 - No bradycardia alarm for 2 months
 - No use of theophylline or caf feine for 2 months
 - e. A normal pneumocardiogram
- Characteristics of the pneumocardi ogram include all but one of the following:

CONTINUED ON PAGE 627

JUNE CME QUIZ **Answers**

Following are the answers to the CME quiz that appeared in the June 1985 issue: "Acquired Immunodeficiency Syndrome: An Overview," by Robert L. Baker, M.D., et al. 1 e

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3.	d					-8.	C
4.						9.	а
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Answer sheet for Quiz: (Apnea . . .)

6. a b c d e 1. a b c d 2. a b c d 7. abcd 3. a b c d 8. a b c d e 1. a b c d 9. a b c d 10. a b c d e 5. a b c d

I wish to apply for one hour of category 1 AMA Continuing Medical Education credit through the I.U. School of Medicine. I have read the article and answered the quiz on the answer sheet above. I understand that my answer sheet will be graded confidentially, at no cost to me, and that notification of my successful completion of the quiz (80% of the questions answered correctly) will be directed to me for my application for the Physician's Recognition Award of the American Medical Association. I also understand that if I do not answer 80% of the questions correctly, I will not be advised of my score but the answers will be published in the next issue of Indiana Medicine.

Name (please print or type)

Address

Identification number (found above your name on mailing label)

Signature

To be eligible for this month's quiz, send your completed, signed application before Aug. 10, 1985 to the address appearing at the top of this page.



AUXILIARY REPORT

Muriel Osborne (Mrs. John) ISMA Auxiliary President 1985-86

On West Michigan Street in Indianapolis, across from the Indiana University Medical Center, stands the "house that love built"—the Ronald McDonald House—a volunteer project that gets considerable attention from the Marion County Auxiliary.

The 24-bedroom "home-away-fromhome" opened to the public Oct. 14, 1982, virtually debt-free, and filled to capacity—the happy result of an all-volunteer undertaking co-ordinated by Our House, Inc., a not-for-profit organization of parents, volunteers, representatives from the Indiana University Medical Center and McDonald's restaurants. The families of patients at Riley Children's Hospital and other Indianapolis area hospitals fill the house to capacity.

The annual report of Lona Damron, Ronald McDonald House manager, shows an occupancy rate of 99.8% for 1984, a statistic that represents worried parents whose children have been stricken with serious illness, more often than not financially strapped and in need of understanding and caring. In addition, Lona's report shows that only slightly more than 50% of the families are able to fully pay for their stay at the house, demonstrating the obvious need for the ongoing financial support of the community and organizations such as ours.

In the spring of 1982, the Marion County Auxiliary contributed \$5,000 toward furnishing two bedrooms, and found itself happily involved in a project that became the rallying point for the membership. The talent and generosity of physicians' spouses are also evident through beautiful needlepoint works of art donated to the House by Jane Beering (Mrs. Steven C.), Drucilla Defalque (Mrs. Ray J.) and Letia Chernish (Mrs. Stanley M.), all prominently displayed on the walls throughout the House.

Ronald McDonald House_™

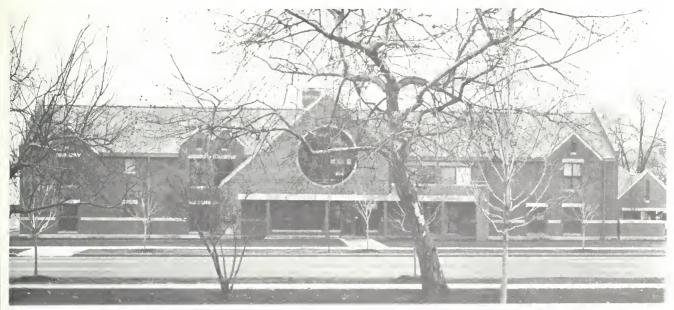
In the summer and fall of that year, the Auxiliary took on the responsibility of furnishing a fully decorated Christmas tree to be displayed in the impressive two-storied greatroom. Project co-ordinator Sally Morton (Mrs. Philip M.) and the membership worked throughout the pre-season months, accumulating hundreds of

crocheted, knitted and hand-painted decorations for the tree, which was officially dedicated in a ceremony at the House Dec. 5, 1982.

Volunteering at the House is a year-round commitment for many auxilians. They work a minimum of two three-hour shifts each month, in addition to other special assignments, and the major reason all of these volunteers are willing to go that extra mile is that each one believes in the philosophy of the Ronald McDonald House—to provide a home-away-from-home for the families of children with serious illnesses. The most important qualification they all possess is that they care!—Anne Throop, Indianapolis, First Vice-President



Inside the Ronald McDonald House.



The Ronald McDonald House in Indianapolis.



Lelia Chernish of Indianapolis (right), a frequent helper at the House, watches as Ronald McDonald prepares to slice a birthday cake.

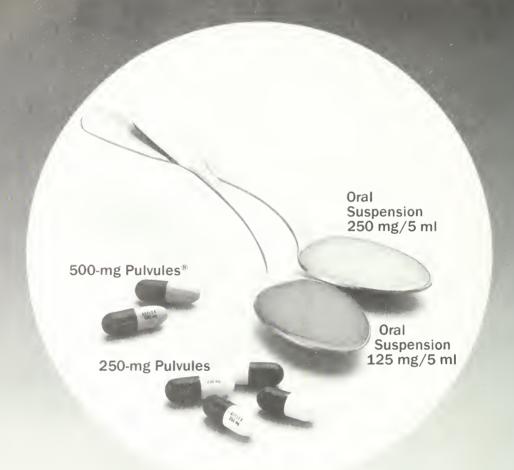


Kenny Flemig, an outpatient at Riley Hospital and a special guest at RMH, pitches in after a holiday celebration.



Christine Barger hugs a "friend" at the Ronald McDonald House.

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NEWS NOTES

New ISMA Members

Richard D. Brown, M.D., Evansville, family practice.

Donn R. Chatham, M.D., New Albany, plastic surgery.

Larry T. Curtis, M.D., Evansville, family practice.

Carol A. Datil, M.D., Evansville, family practice.

James W. Edmondson, M.D., Indianapolis, endocrinology.

Gordon R. Franke, M.D., Fort Wayne, family practice.

Louis A. Gluek III, M.D., Munster, orthopedic surgery.

Darla R. Grossman, M.D., Evansville, family practice.

Jeanne L. Grossnickle, M.D., Goshen, diagnostic radiology.

Jan Gullberg, M.D., Angola, general surgery.

Rick L. Hoover, M.D., South Bend, pathology.

Matthew R. Lee, M.D., Mount Vernon, family practice.

Charles X. McCalla IV, M.D., Marion, emergency medicine.

Virginia E. Mrizek, M.D., Hammond, family practice.

Jerome A. Olack, M.D., Marion, radiology.

Hypertriglyceridemia

CONTINUED FROM PAGE 609

disease, etc. which can be associated with increased incidence of cardiovascular disease.

With levels greater than 500 mg./dl there is increased risk of pancreatitis. Diet and/or drugs is mandatory to lower the levels of triglycerides.

The primary approach for elevated triglycerides is diet, with reduction of saturated fat and associated exercise. Up to a year may be necessary to successfully lower triglycerides through diet. Drugs should be used only in selected nonresponders and stopped if the drugs fail to help.

More research is needed in the area of elevated plasma triglycerides to better understand the association of atherogenesis, and improve approaches to treatment.—I. E. Michael, M.D., Indianapolis

Jeffrey L. Payne, M.D., Owensboro, Ky., family practice.

James B. Records, M.D., Indianapolis, family practice.

D. Peter Reedy, M.D., Lafayette, neurological surgery.

R. Kevin Rogers, M.D., Evansville, family practice.

Paul W. Tittel, M.D., Charlestown, diagnostic radiology.

Mark A. Truax, M.D., Crawfords-ville, family practice.

David R. Ware, M.D., Bedford, family practice.

Michael P. Yoder, M.D., Goshen, family practice.

A Solution for Physicians Wanting to Relocate

The American Academy of Family Physicians (AAFP) has announced a new service to help put family physicians in contact with practice opportunities.

Computer-assisted Site Selection (COMPASS) will act as a clearing house and will exchange information between family physicians looking for a practice site and the many types of persons such as other physicians, communities, hospitals and others who are seeking a physician to join, buy or start a practice.

The service will operate nationally. It has a fee schedule for various categories of people on both sides of the exchange.

COMPASS was tested in early 1985 in Missouri and Kansas with encouraging results.



"Let me put it this way—if you were a huilding you'd he past restorinig."

\$45 Million Committed for Nucleic Acid Research

A joint research institute that will explore new biomedical compounds aimed at stopping the spread of viral infection and slowing the aging process has been formed by Eastman Kodak Company and ICN Pharmaceuticals.

Kodak and ICN will invest \$45 million over a period of six years to form and operate the Nucleic Acid Research Institute, a joint venture that will be located at ICU's facility in Costa Mesa, Calif. The institute will dedicate much of its research exclusively to preclinical studies of new antiviral and anti-aging substances.

Convention Calendar

The following dates and sites have been established for future ISMA annual conventions:

1985: Nov. 14-17, South Bend Century Center and Marriott.

1986: Oct. 30-Nov. 2, Hyatt Regency, Indianapolis.

1987: Nov. 5-8, Radisson Hotel, Indianapolis.

1988: Nov. 3-6, Hilton Inn, Indianapolis.

1989: Nov. 2-5, Indianapolis (site not chosen).

1990: Nov. 1-4, Indianapolis (site not chosen).

CME Quiz

CONTINUED FROM PAGE 623

- a. In some patient populations, (e.g., premature infants) it may be used as a screening tool to identify risk for SIDS
- b. It is a 2 channel recording of cardiorespiratory variables
- c. The recording may be obtained in the home or hospital setting
- d. It is obtained using a monitor based on impedance pneumography
- 10. Prolonged apnea can be the initial symptom of:
 - a. sensis
 - b. bronchiolitis
 - c. apnea of infancy
 - d. meningitis
 - e. all of the above

NEWS NOTES

Here and There . . .

Dr. Robert T. Woodburn, a Merrillville hematologist, is a new fellow of the American College of Physicians.

Dr. Robert J. Voorhees has been named medical director of the burn unit at St. Joseph's Hospital, Fort Wayne.

Dr. Robert M. Lohman is the new president of the medical staff at Lutheran Hospital, Fort Wayne.

Dr. J. F. Hinchman of Parker City is a new member of the board of directors, Parker Banking Co.



Dr. Grosfeld

Dr. Jay L. Grosfeld, director of pediatric surgery, Riley Hospital for Children since 1981, has been appointed chairman of the Department of Surgery, I.U. School of medicine.

Dr. Arvine G. Popplewell was honored posthumously by being named "Manual 1985 Alumnus of the Year" at graduation exercises in May. He was a 1941 graduate of Manual High School, Indianapolis.

Dr. Clarence E. Ehrlich of Indianapolis is the new secretary-treasurer of the Indiana Section, American College of Obstetricians and Gynecologists.

Dr. Richard T. Miyamoto of Indianapolis discussed "Cochlear Implants" at the recent international conference at the Inter-University center in Dubrovnik, Yugoslavia.

Dr. Harry T. Hensley of Oaklandon has been named medical director of Pleasant View Lodge, a Hancock County nursing home.

Dr. Hansel O. Foley is the new med ical staff president at Memorial Hospital, South Bend; Dr. Alfred C. Cox is vice-president, and Dr. James L. Grainger is secretary-treasurer.

Dr. Robert J. Burkle, a Terre Haute orthopedic surgeon, has received the Wellmerling Award from the American Fracture Association.

Dr. John E. Mackey of Indianapolis, clinical professor of Ob/Gyn at Indiana University, has been presented the Edwin L. Gresham Award by the Indiana Chapter, American Academy of Pediatrics, for contributions in perinatal medicine.

Dr. John U. Keating, a psychiatrist at the Logansport State Hospital, has retired after serving 17 years with the Indiana Department of Mental Health.

Dr. Stephen E. Braun is the new medical staff president at Deaconess Hospital, Evansville; Dr. David J. Carlson is president-elect, and Dr. William F. Johnson is secretary-treasurer.

Dr. R. Joe Noble of Indianapolis has been appointed to the Subspecialty Board of Cardiovascular Disease, American Board of Internal Medicine.

Dr. Keven W. Dodt of Logansport participated in a panel discussion on child abuse, sponsored in May by Memorial Hospital.

Dr. Steven C. Meyer of Fort Wayne addressed Warsaw's Home Health Care-Hospice volunteers in May.

Dr. Joe G. Conley, Dr. Aftab Chaudhry and Dr. Howard A. Pope of New Albany served on a panel in May to answer viewer call-in questions following a TV presentation of "Cancer Today."

Dr. Paul D. Isenberg of Indianapolis discussed allergies and asthma at a May meeting sponsored by St. Francis Hospital.

Dr. Samuel M. Wentworth of Danville presented the keynote address at the May meeting of the Vigo County Chapter, American Diabetes Association.



Dr. Bull

Dr. Marilyn J. Bull, director of the Newborn Follow-up Program at Riley Hospital for Children, has won the Irving Rosenbaum Award for Community Service from the Indiana Chapter, American Academy of Pediatrics.

Dr. Eric G. Friedman of Valparaiso was guest speaker at the May meeting of the Porter County Chapter, American Diabetes Association.

Dr. James J. Laughlin of Bloomington discussed pediatrics at a May meeting of the Local Council of Women.

Dr. Michael J. Roselman of Evansville was guest speaker at a May meeting of the Greater Evansville Chapter, Data Processing Management Association.

Dr. Michael S. McCrea of Terre Haute discussed breast cancer during a May symposium sponsored by Vermillion County Hospital.

Dr. Stephen W. Perkins of Indianapolis recently directed a two-day seminar at Indiana University on facelift, liposuction, hair replacement and tissue expanders in the head and neck.

Dr. John L. Jenkins, a South Bend cardiologist, addressed a May meeting of the local Mended Hearts Club.

Dr. Joseph C. Sheehy of Columbus discussed exercise and the heart at a recent meeting of the Heart Beats Club.

Dr. David B. Goldenberg of Indianapolis discussed breast disease and early detection during a May Heartbeats Health Festival at the Hancock County Fairgrounds.

Dr. Charles M. Clark Jr., director of the diabetes research and training center at Indiana University Medical Center, has been appointed to the National Diabetes Advisory Board.

Dr. William F. Nowlin of Merrillville discussed cancer research during a May American Cancer Society seminar in Merrillville.

Dr. Lynn E. Eiler of Lawrenceburg discussed diabetes and heart disease at a recent meeting at Dearborn County Hospital of the Heartbeats Club. Dr. II. Michael Mann of Indianapolis discussed the effect of anesthesia on patients with chronic respiratory problems during a May meeting of the Johnson County Respiratory Health Club.

Dr. William G. Terpstra of Noblesville discussed headaches during a May presentation sponsored by Riverview Hospital.

Dr. Charles G. Griffin of Valparaiso participated in a recent symposium on the medical and legal aspects of death and dving.

Dr. Jose C. Torres of Jeffersonville discussed gastric bypass procedures at a recent meeting in Louisville of the Kentucky Chapter, American College of Surgeons.



"You have a lot in common with my last doctor—he didn't have any faith in my self-diagnosis either."

Physician Recognition Awards -



The following ISMA physicians are recent recipients of the AMA's Physician Recognition Award. This award is official documentation of Continuing Medical Education hours earned, and is acceptable proof in most states requiring CME in re-registration that the mandatory hours of CME have been accomplished.



Alexander, Panos C., Kokomo Anderson, Milton H., Evansville Ayoub, Adel H., Valparaiso Beaver, Steven R., Rensselaer Bradenberger, E. Jon, Fort Wayne Buck, Richard C., South Bend Chua, Felipe S., Merrillville Cole, Stephen L., Auburn Conley, John E., Indianapolis Cortese, Thomas A., Indianapolis Darbro, David A., Indianapolis Dauscher, Dean D., Fort Wayne Diaz, John R., Indianapolis Eberts, Thomas J., South Bend Ebbinghouse, Tom H., Richmond Echsner, Herman J., Columbus Eckert, Russell A., Logansport Eller, Alvan L., Flora Farag, Rafik S., Peru Fretz, Richard C., Kokonio Goldschmidt, Max W., Munster Goldsmith, David A., Marion Gordon, Mark, Munster

Greenwood, Charles W., Columbus Hamm, Charles W., Indianapolis Heaton, Elton, Madison Haynes, John T., Indianapolis Holm, Byron M., Jr., Plymouth Horton, Douglas J., Indianapolis Kelley, William E., Mooresville Kincaid, Raymond K., Tipton Krol, John E., South Bend Lautz, Herbert A., Munster Lyons, Charles R., Wabash Mayer, John R., Indianapolis McFadden, Wilbur D., N. Manchester Michl, Leon G., Madison Murray, Richard P., Evansville Miller, L.H., Indianapolis Miller, Thomas P., Indianapolis Monn, Larry N., Carmel Moore, Thomas S., Indianapolis Nallinger, Richard E., Madison Neathamer, Thos. A., Jeffersonville Paff, James R., Kokomo

Pantzer, John G., Indianapolis Raber, Robert M., Indianapolis Rice, Frederic A., Indianapolis Roth, James R., Wolflake Rouhana, Rodolph, Indianapolis Sechrist, Keeter D., Indianapolis Shanklin, Jack L., Vincennes Sklenarz, Krystyna M., Merrillville Sheehy, Joseph C., Columbus Sinkovic, Gerald M., Indianapolis Smith, Jeffery, Portage Spalding, David L., Mishawaka Sprecher, James J., LaPorte Stone, Dennis E., Columbus Subhasiriwat, Man, Crown Point Underwood, George M., Lafayette Volan, George J., Merrillville Waiss, Elaine H., Munster Wayne, Lisle, II, Evansville Webb, Orville L., New Castle Webb, Thomas A., Evansville Zollman, Charles W., Indianapolis

NEWS NOTES

For the Asking . . .

- The American Porphyria Foundation, recently formed, will, upon request, provide general information, research updates and sources of expert testing and medical advice. Address: P.O. Box 11163, Montgomery, Alabama 36111.
- A 22-minute videotape, "The Breast: A Realistic Guide to Self-examination and Mammography," gives doctors a consistent and direct method of educating patients in their office (or the tape may be taken home by the patient). For a detailed brochure, write: Apogee Communications Group, 382 Alpine Way, Boulder, Colo. 80302
- "What Are Clinical Trials All About?" is the title of a booklet published by the National Cancer Institute. It explains the nature of clinical trials in lay language and supplements the information patients receive from health professionals. Presented in Q & A style, it answers such questions as What is a clinical trial? Why are clinical trials important? How are they conducted? Are there potential benefits and potential risks in trials? What are the unknowns? Physicians may obtain copies in numbers of 1, 50, 100 or 200. Contact: Office of Cancer Communications, National Cancer Institute, Bldg. 31, Rm. 10A18, Bethesda, Md. 20205 - 1-800-4-CANCER.
- A Louis Harris and Associates survey on 1984 attitudes and experiences with HMOs has been released. This is the fourth such survey by Harris, all of which were commis sioned by the Henry J. Kaiser Family Foundation. The latest survey presents interesting changes in attitudes since the earlier surveys in 1980 and 1981. The present survey is titled "A Report Card on HMOs: 1980-1984." Copies of the full report, copies of a summary of the findings, or information about obtaining the data tape are available from the Henry J. Kaiser Family Foundation, 525 Middlefield Road, Suite 200, Menlo Park, Calif. 94025.

- "Kodak Products for Special Biomedical-Imaging Applications" is a recently expanded catalog that provides information about macro-autoradiography, electron micrography, processing and safelighting. It includes new information on products for photomicrography, making lecture slides, and CRT photography. For a free copy of Catalog M6-1, contact any Kodak regional marketing center or write Eastman Kodak, Dept. 412-L, 343 State St., Rochester, N.Y. 14650.
- "Power Lawnmowers Are Dangerous to Children" is a pamphlet produced by Methodist Hospital of Indiana based on its own experience and on data from the U.S. Consumer Product Safety Commission. Up to 20 copies of the pamphlet, aimed at parents, are available free of charge. They may be ordered in multiples of 50 at 5° each. Write Pediatric Medical Education, Methodist Hospital, P.O. Box 1367, Indianapolis, Ind. 46206.

Computer Medicine Association Formed

The American Physicians Association of Computer Medicine, Inc. has been formed as a non-profit organization dedicated to encouraging and educating physicians in the use of computers in medical practice. Membership is limited to licensed physicians and osteopaths.

APACM is an educational organization that will coordinate the development of computer programs for patient care, disseminate information on existing programs, establish a medical software library, and develop guidelines and standards for computer use in direct patient care relating to the security of patient medical data and liability for programs used in patient diagnosis and treatments.

For more information write to the association at 10 N. Main St., Pittsford, N.Y. 14534.

Interferon Update

Interferon is an immunomodulating agent that may be of value in treating both viral infections and cancer. Several interferons are now available for research projects, thanks to the ingenuity of genetic engineers.

It has been learned that, in addition to inducing a state of resistance to viruses in cells, genetic engineers also have been able to slow the rate of cell multiplication. To date, we know of three antigenically distinct types of interferons named after their cell of origin: "leukocyte," "fibroblast" and "T cell" (or "immune") interferons. These three types are now designated as Alpha, Beta and Gamma, respectively.

As practical information is published about this exciting group of compounds, more information will be provided.—I.E. Michael, M.D., Indianapolis

\$20,000 Fellowship Awards

The deadline to apply for five 1986-87 Adult Cardiology Fellowship Training Awards is Dec. 1, 1985. These are the fellowships offered by the American College of Cardiology and The Merck Company Foundation. Each fellowship provides a \$20,000 stipend and an additional \$5,000 for the purchase of supplies and equipment to carry out the project. More information from: ACC, Membership Services Dept., 9111 Old Georgetown Road, Bethesda, Md. 20814.

Cochlear Implant Cleared

A long-term study to evaluate the physiological impact of the cochlear implant in patients who suffered from profound hearing loss shows that the implanted House-Urban electrode does not cause osteoneogenesis (new bone growth) in the cochlea. Dr. Richard Miyamoto, I. U. Medical Center, who did pioneering work on the device, reported the long-term reactions at a recent meeting of the American Neurotology Society.

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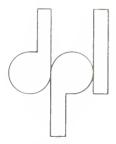
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OBITUARIES

Louis E. How, M.D.

Dr. How, 84, a retired Lakeville general practitioner, died April 17 at a local retirement home.

He was a 1924 graduate of General Medical College, Chicago. He retired in 1971 after a 17-year tenure as St. Joseph County health officer.

Dr. How, a former president of the 13th District Medical Society, was named physician of the year in 1966 by the St. Joseph County Mental Health Department and, in 1975, he was the recipient of the Jaycees' Good Government Award. He was a member of the ISMA Fifty Year Club.

Henry J. Rusche, M.D.

Dr. Rusche, 63, an Evansville general practitioner, died May 9 at Deaconess Hospital.

He was a 1946 graduate of Indiana University School of Medicine and was a veteran of the Korean War.

Dr. Rusche was a former president of the Deaconess Hospital medical staff. He was a member of the American Academy of Family Physicians.

Leon J. Garrison, M.D.

Dr. Garrison, 85, a retired Gas City (Grant County) general practitioner, died April 7 at his home.

He was a 1930 graduate of Indiana University School of Medicine. He retired in 1968.

Dr. Garrison was a member of the ISMA Fifty Year Club.

Charles W. Myers, M.D.

Dr. Myers, 94, a retired Indianapolis physician who formerly served as superintendent and medical director of the predecessors of Wishard Memorial Hospital, died May 13 at his home.

He was a 1915 graduate of the University of Maryland School of Medicine and was an Army veteran of World War I, during which he won the Distinguished Service Cross.

From 1952 to 1968 Dr. Myers was a trustee of the Health and Hospital Corp. He was a fellow of the American College of Surgeons and was a former president of the Indiana Hospital Association. He became a member of the ISMA Fifty Year Club in 1965.

Richard M. LaSalle, M.D.

Dr. LaSalle, 53, a Wabash general practitioner, died April 5 at Wabash County Hospital.

He was a 1956 graduate of Indiana University School of Medicine.

Dr. LaSalle was a former president of the Wabash County Medical Society. He also was a former vice-president of the Wabash County Hospital medical staff and a former county coroner. He was a diplomate of the American Board of Family Practice and was a member of the American Academy of Family Physicians and the American Society of Abdominal Surgeons.

MA Fifty Year Club. Surgeons.

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Isadore J. Kwitny, M.D.

Dr. Kwitny, 82, executive director of the Medical Licensing Board of Indiana until 1981, died May 30 at his home in Indianapolis.

He was a 1928 graduate of Indiana University Schoot of Medicine, where he tater taught for 25 years in addition to his private practice. He was an Army veteran of World War II.

Dr. Kwitny, a former president of the medical staff at St. Vincent Hospital, retired from medical practice in 1977. He was a diplomate of the American Board of Internal Medicine and was a member of the ISMA Fifty Year Club.

George E. Oldag, M.D.

Dr. Oldag, 68, a retired Elwood (Madison County) surgeon, died April 21 at a local nursing home.

He was a 1942 graduate of the University of Iowa College of Medicine.

Dr. Oldag was a diplomate of the American Board of Surgery and a fellow of the American College of Surgeons.

Hubert M. English, M.D.

Dr. English, 94, a retired Gary general practitioner, died May 12 in an Illinois nursing home.

He was a 1918 graduate of Harvard Medical School and was an Army veteran of World War I.

Dr. English, who retired in 1971, was a past president of the Lake County Medical Society and of the Gary Board of Health. He was also a former director of the Gary Chapter, American Red Cross and was a member of the ISMA Fifty Year Club.

Charles L. Entner, M.D.

Dr. Entner, 83, a retired Dunkirk general practitioner, died May 1 at Jay County Hospital.

He was a 1934 graduate of Tufts College of Medicine, Boston.

Dr. Entner, who retired in 1980, spent nine years in Nigeria as a medical missionary.

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Cantraindicatians: Known hypersensitivity to flurozepom HCI, pregnancy Benzodiozepines moy cause fetal damage when administered during pregnancy Several studies suggest on increased risk of congenital multormations associated with benzodiozepine use during the first trimester. Worn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurozepom. Instruct potient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting theropy.

Warnings: Caution patients obout possible combined effects with olcohol and other CNS depressants. An additive effect may occur if olcohol is consumed the day following use for nightime sedation. This potential may exist for several days tollowing discantinuation. Coution against hozardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported an recommended doses, obrupt discantinuation should be avaided with gradual tapering of dosage for those potents on medication for a pralanged period of time. Use coution in administering to addiction-prone individuals or those who might increase dosage.

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Adverse Reactians: Dizziness, drowsiness, lightheadedness, stoggering, otoxio and falling have accurred, porticularly in elderly or debilitated patients. Severe sedation, lethargy, disarientation and como, probably indicative at drug intalerance or overdosoge, have been reparted. Also reported headoche, hearthurn, upset starmoch, nouseo, vormiting, diarrhea, canstipotian, Glipoin, nervousness, talkativeness, opprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU camploints. There have also been rare accurrences of leukopenia, granulacytapenia, sweating, tlushes, difficulty in focusing, blurred vision, burning eyes, taintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter toste, excessive salivatian, anarexia, euphoria, depression, slurred speech, contusion, restlessness, hollucinations, and elevoted SGOT, SGPT, total and direct bilirubins, and olkaline phosphatase, and paradaxical reactians, e.g., excitement, stimulation and hyperoctivity

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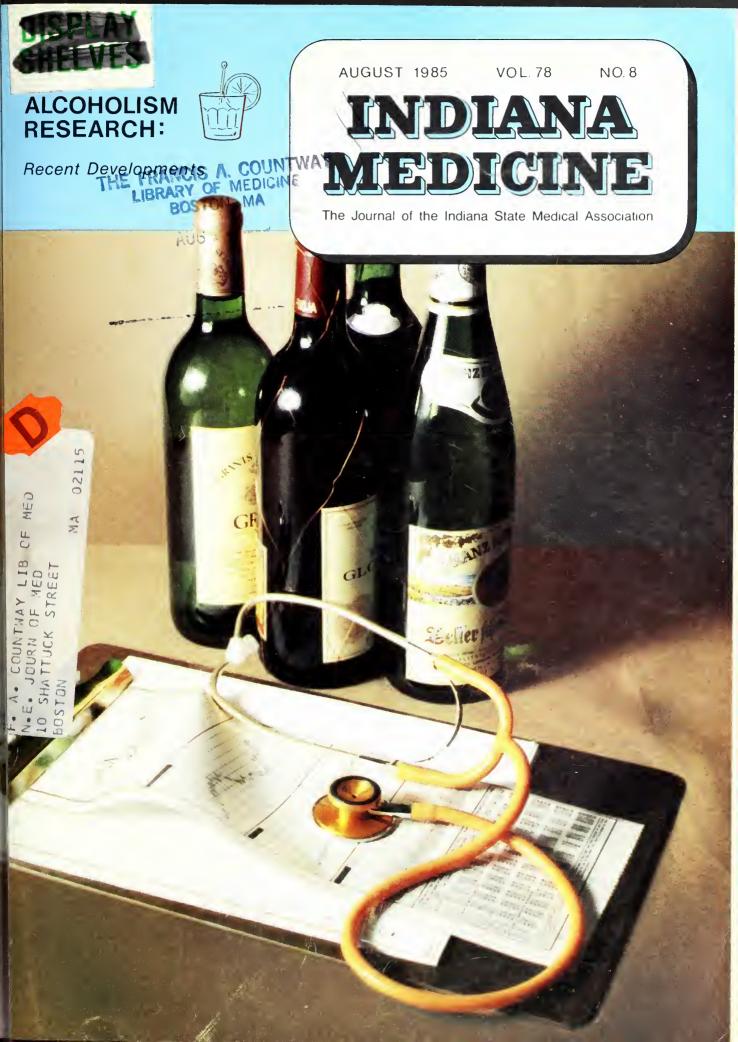
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ABOUT THE COVER

An estimated 7% of the adult American population drink to ex cess. This month's Continuing Medical Education installment. prepared by two Indiana University School of Medicine physicians, deals with recent developments in alcoholism research. INDIANA MEDICINE wishes to thank Jeanne Perkins of Koala Centers for the cover concept and Gary W. Potts for the photography.

CONSULTING EDITORS

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MEDICAL MUSEUM NOTES

CHARLES A. BONSETT, M.D., Indianapolis



ment of the U.S. Geological Survey map of the Chief Mountain quadrangle (Montana) to show the location of Mount Wynn, named for Hoosier physician Frank B. Wynn.

William J. Briggle, who was superintendent of Glacier National Park in 1971 when I first inquired about the mountain's location, wrote as follows:

"... Mt. Wynn was named for Frank B. Wynn, physician and scientist who was killed in an attempt to climb Mt. Siyeh on July 27, 1922. This mountain was originally named 'Point Mountain' by George Bird Grinnell on his map of 1885-92. Later the miners from the mining town of Altyn, near the head of Sherburne Lake, named it Altyn after their town, and in 1923 topographers transferred this name to a mountain overlooking Swiftcurrent where it remains, and renamed the former mountain 'Mt. Wynn.'"

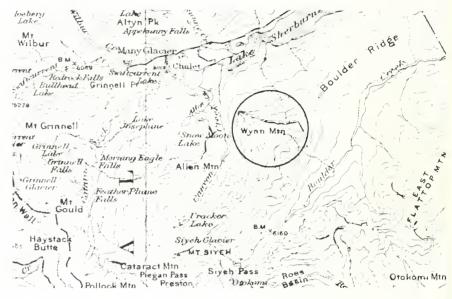
Dr. William B. Niles Jr. sent the following note in January 1973, along with a yellowed newspaper clipping:

"I came across the enclosure today and thought you might like to have it. It's an editorial from the (*Indianapolis*) *News*, probably 1923."

Honoring Dr. Wynn

"Friends of Dr. Frank B. Wynn—and they were numbered by thousands—will be glad to know that a mountain peak in Glacier National Park is to bear his name. This peak, designated by the National Geographic Board, rises impressively at the mouth of Canyon Creek, where one of the most used trails of the glacier region winds to Cracker Lake.

"Dr. Wynn had climbed mountains in Switzerland, Canada and this country for more than 25 years. He had served as president of the American Alpine Club and had cooperated with the Department of the Interior in mapping trails that less venturesome climbers might use during their excursions in Glacier National Park. Some of his climbing feats, notably his effort



to scale Mt. St. Wilbur, have not been duplicated. He died last year while climbing from Piegan Pass up the sides of Mt. Siyeh, one of the Glacier Park peaks.

"Nothing could be more fitting as a lasting memorial to the intrepid nature of Dr. Wynn than the action that the government has taken. He loved nature as few love it, and it is especially appropriate that one of nature's monuments should bear his name. Dr. Wynn found companionship in mountains because he was like them. His spirit soared beyond the peaks and his beliefs were as well grounded as the foundations from which the mountains tower. The conservation commission truthfully said of him that it sought only to acknowledge 'the Commonwealth's vast debt to one of her foremost sons, who, clinging to his state with all the fibers of a pure heart and a lofty mind, representing by tradition and inheritance the debt of a great formative past, brought health, happiness and understanding through the skill of his profession and the magic of his soul to those he knew and loved best."

In 1919, Dr. Wynn was the first to ascend Going-to-the-Sun Mountain, where he left this original poem in a cairn at the peak to commemorate the event:

MOUNTAIN-TOP PRAYER

Dear Lord, I thank Thee for this view Of paradise.

The fearsome trail was hard to do, But worth the price.

The arching canopy of art In Heaven wrought,

Encompasses the very heart Of beauteous thought.

In Nature's lap of forest green, Rests tranquilly

The shimmering lake; the glinting stream

Leaps jously.

The serried ranks of snow-clad peaks Attention stand,

Like faithful, white-robed sheiks Await command.

Thy handiwork! How wondrous and How beautiful!

My soul enraptured bids my hand Be dutiful!

The trudging up you toilsome trail How well repaid!

'Twill help me in life's sore travail, Hath courage made!

For strength of limb and will to do And try again,

I thank Thee, Lord, and pledge anew My faith. Amen!

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Foods that may help reduce the risk of gastrointestinal and respiratory tract cancer are cabbage, broccoli, brussels sprouts, kohlrabi, cauliflower.

> Fruits, vegetables and wholegrain cereals such as oatmeal, bran and wheat

may help lower the risk of colorectal cancer.

Foods high in fats, salt- or nitrite cured foods such as ham, and fish and types of

sausages smoked by traditional methods should be eaten in moderation

Be moderate in consumption of alcohol also

A good rule of thumb is cut down on fat and don't be fat Weight reduction may lower cancer risk. Our 12 year study of nearly a million Americans uncovered high cancer risks particutarty among people 40% or more overweight

Now, more than ever, we know you can cook up your own defense against cancer. So eat healthy and be healthy

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MHAT'S NEWS

General Electric has a new obstetrical ultrasound imaging system. It weighs just over 12 pounds. The highly portable RT50TM System al lows obstetricians and other physicians to obtain high quality images from the patient's first examination through delivery. It is suitable for use in the office, in the hospital room and in the delivery room.

BNA Communications is distributing a new film/video that provides analysis of labor unions' new survival tactics. It is available for license or rental. The film focuses on the forces and problems facing unions today and how specific unions are confronting their declining power and influence. A blend of commentary, case studies. and interviews with noted labor relations experts is employed to provide understanding of a complex topic.

American Health Consultants, an Atlanta-based health care communications company, has published the first major reference text for setting up or operating a clinical laser program. Clinical Lasers: Expert Strategies for Practical and Profitable Management is the title. It discusses applications for the laser-using surgical specialties and examines the anesthesia risks with asers. Various forms, checklists. worksheets and sources of information appear in five appendices, 356 pages, \$128.



News of what is new in the medical supply industry is composed of abstracts from news releases by book publishers and manufacturers of pharmaceuticals, clinical laboratory supplies, instruments and surgical appliances. Each item is published as news and does not neces sarily constitute an endorsement of a product or recommendation for its use by Indiana Medicine or by the Indiana State Medical Association.

Western Enterprises has a new Oxygen Analyzer which detects oxygen purity in seconds. Employing advanced microprocessor technology, the new analyzer uses an LCD readout to display the per cent of oxygen purity in oxygen cylinders.

The new, fully cumulative 1986 edition of USAN and the USP Dictionary of Drug Names is being published by the United States Pharmacopeial Convention. It is the intent of the FDA that the U.S. Adopted Names (USAN) and the compendial (USP and NF) names comprise the "established names." Only those names constitute the main list and are printed in boldface type.

Searle announces FDA approval for labeling indications for intravenous Flagyl[®] as a prophylaxis for elective colon and rectal surgery. After thorough bowel cleansing patients were either given Flagyl I.V. and oral neomyein or placebo and oral neomycin. The first group remained infection-free. The second group had an infection rate of 21%. The dosage of Flagyl I.V. was 15 mg/kg body weight one hour before surgery, and 7.5 mg/kg body weight I.V. at six and 12 hours after surgery. Neomycin was given in four one-gram doses orally once every four hours on the day before surgery. Cost containment was improved by a shorter preparation time, less infection after operation and a shorter hospital stay after operation.

Unimed, Inc. announces FDA approval for marketing Marinol (dronabinol) for the alleviation of severe nausea and vomiting associated with cancer chemotherapy.

Abbott Laboratories is marketing an ambulatory medication infusion pump developed by Parker Hannifin Corporation. The programmable electronic infusion device will be used to provide drugs to patients being treated for cancer, pain and infectious diseases. Since the device is ideal for out-patient use, it will reduce hospital costs. Known as the LifeCare 1500 ambulatory microinfuser, it is the most sophisticated and the most compact on the market.

Amko announces a heavy-duty Tischler biopsy punch (G-642). It is made of stainless steel, 9" long, and is the latest addition to Amko's complete line of Eppendorfer, Kevorkian and Wittner biopsy punches.

General Electric announces a new infant positioner for supine studies in the radiology lab. It will comfortably immobilize children up to age 3 for supine diagnostic imaging. Retakes are reduced and diagnostic images are improved.

Abbott Laboratories has a new, fast, accurate, easy-to-use desk top blood analyzer that is expected to rapidly accelerate growth in the market for physicians' office testing. It is fully automated, its name is Vision, and it produces results in eight minutes from whole blood.



you in bed.

FUTURE FILE

Indiana University CME

For the Primary Care Physician

Aug. 14—Infectious Disease Update, Indianapolis.

Sept. 25 - Practical Management Protocols in Office Gynecology, Indianapolis.

Date Negotiable — Mini-Fellowship in Rheumatology. (For details, see IN DIANA MEDICINE, p. 448, June 1985).

For the Specialist

Sept. 27-28 – Management of the Patient with Breast Cancer, Indianapolis.

Sept. 30-Oct. 2 – Advanced Echocardiography.

For more information, contact the CME Division, I.U. School of Medicine – (317) 264-8353.

St. Vincent CME

Sept. 4-5: 10th Annual Arthur B. Richter Lectureship in Clinical Cardiology (H. Jeremy Swan, M.D., Los Angeles, guest lecturer), St. Vincent Hospital, Indianapolis.

Sept. 25: Contemporary Clinical Nutrition Update, Ritz Charles Conference Center, Indianapolis.

Nov. 13: 4th Annual Symposium on Ethical and Moral Issues in Medicine, Holiday Inn North, Indianapolis.

Nov. 18-19: Institutional Review Board Conference/Ethics and Regulation of Clinical Research, Hyatt Regency, Indianapolis.

For additional information contact Marilyn Soltermann, CME coordinator, St. Vincent Hospital and Health Care Center – (317) 871-3460.

Focus on Rheumatology

"Focus on Rheumatology — 1985" is the title of a CME program being promoted by the University of Wisconsin. The course will be conducted in the U. of W. Clinical Science Center Oct. 18 and 19. It is designed for physicians, nurses and other health professionals, and offers 8½ hours of AMA Category 1 and AAFP credit.

Contact Sarah Aslakson, 465B WARF Bldg., 610 Walnut St., Madison, Wisc. 53705-(608) 263-2856.

The Journal of the American Medical Association publishes a list of CME courses for the United States twice yearly. The January listing features courses offered from March through August; the July listing features courses offered from September through February.

Hospital Privileges

"Hospital Privileges and Specialty Medicine" is the topic of a joint conference of the American Board of Medical Specialties and the American Hospital Association, to be conducted Sept. 17 and 18 at the Marriott Hotel O'Hare, Chicago.

Registration for ABMS members and for AHA personal and institutional members is \$200. The fee for non-members is \$300.

Write ABMS, One American Plaza, Suite 805, Evanston, Ill. 60201.

PerIstein Lecture

The annual John I. Perlstein Lectureship will be presented Monday, Aug. 26, by the Dept. of Pediatrics, University of Louisville School of Medicine, in the Health Sciences Center Auditorium, Abraham Flexner Way.

Dr. Margaret H. D. Smith, clinical professor of pediatrics at Tulane University School of Medicine, will lecture on "Diseases of Animals Transmitted to Children," beginning at 11 a.m.

Dr. Smith is internationally recognized for her numerous contributions to pediatrics and the understanding and management of infectious diseases through her research, publications, presentations, and service to many national and world organizations.

All practicing physicians, house staff, medical students, other allied health personnel, and all those interested are invited to the lecture.

For more information, contact Billy F. Andrews, M.D., Dept. of Pediatrics, University of Louisville School of Medicine, Louisville, Ky. 40292.

Practice Opportunity Fair

A Practice Opportunity Fair for resident physicians and physicians desiring to relocate will be conducted Friday, Sept. 6, from 4:30 to 8:30 p.m. at the I.U. Medical Center.

Displays and tables set up by hospitals and communities seeking physicians, as well as physicians seeking to expand their practice, will be included. The fair is sponsored by the ISMA Resident Medical Society and the IUMC Housestaff Association.

To reserve table space or to receive more information, contact Carol Ann Cunningham at ISMA headquarters, Indianapolis.

Eating Disorders

"Eating Disorders" is the topic of a CME meeting scheduled by the University of Wisconsin at Madison. It will be conducted Oct. 11 and 12 at the Westowner in Madison. Ten hours of AMA Category 1 credit has been assigned, and Family Practice credit is pending.

Contact Sarah Aslakson, 465B WARF Bldg., 610 Walnut St., Madison, Wisc. 53705 – (608) 263-2856.

Methodist Hospital CME

Oct. 2: 10th Fred Priebe Symposium on Rheumatoid Arthritis, Methodist Hospital, Indianapolis.

Oct. 4-5: Advanced Trauma Life Support, Methodist Hospital, Indianapolis.

Oct. 16: Midwest Neuro Critical Care Seminar III, Methodist Hospital, Indianapolis.

Oct. 17-20: 4th National Seminar on Community Cancer Care, Hyatt Regency, Indianapolis.

Oct. 23-24: Fall Wishard Lecture, Methodist Hospital, Indianapolis.

Oct. 29-30: 4th Annual Pediatric Critical Care Symposium, Radisson Plaza, Indianapolis.

For more information, contact Dixie Mattingly, CME coordinator, Graduate Medical Center, Methodist Hospital of Indiana—(317) 929-3733.

CONTINUED ON PAGE 707

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CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

References

- Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. <u>Am J Cardiol</u> 49:560-566, 1982.
- Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exerciseinducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

Reduces angina attack frequency* 42% to 46% decrease reported in

multicenter study.

Increases exercise tolerance*

In Bruce exercise test, control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes (P<.005).

CARDIZEM

(diltiazem HCl)

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PROFESSIONAL USE INFORMATION



DESCRIPTION

CARDIZEM* (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy) -5-12-(dimethylamino)ethyl]-2,3-dihydro-2-14-methoxyphenyl)-. monohydrochforide.(+)-cis- The chemical structure is

Diltiazem hydrochloride is a white to off-white crystalfine powder with a bitter taste It is soluble in water, methanol, and chloroform It has a molecular weight of 450 98 Each table of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth

Mechanisms of Action. Although precise mechanisms of its

antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways

1 Angina Due to Coronary Artery Spasm CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial

and subendocardial Spontaneous and ergonovine-induced cor-onary artery spasm are inhibited by CARDIZEM 2 Exertional Angina CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal

and maximal exercise work loads
In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause fittle or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in sys-temic blood pressure and decreases in peripheral resistance

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, dilliazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect, cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltrazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem

Intravenous diffragem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus

cycle length (up to 50% in some cases)
Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients

Pharmacokinetics and Metabolism. Dilitazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40% CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl dilitiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem Therapeutic blood levels of CARDIZEM appear to he in the range of 50 to 200 ng/ml There is a departure from dose-linearity when single doses above 60 mg are given, a 120-mg dose gave blood levels three times that of the 60-mg dose There is no information about the effect of renal or hepatic impairment on excretion or metaholism of diltiazem

INDICATIONS AND USAGE

Angina Pectoris Due to Coronary Artery Spasm. CARDtZEM

is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment

efevation occurring during attacks)
Chronic Stable Angina (Classic Effort-Associated Angina). CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in

reducing angina frequency and increasing exercise toferance. There are no controlled studies of the effectiveness of the concomitant use of dilitazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) natients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

Cardiac Conduction. CARDIZEM prolongs AV node refrac tory periods without significantly prolonging sinus node ery time, except in nationts with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0 48%). Concomitant use of diltrazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem

2 Congestive Heart Fallure. Although diltiazem has a negative

inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.

3 Hynotensian Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hynotension

Acute Hepatic Injury. In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General, CARDIZEM (diltrazem hydrochloride) is extensively metabofized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods. Jaboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of dittiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs doses of 20 mg/kg were also associated with henatic changes

however, these changes were reversible with continued dosing **Drug Interaction.** Pharmacologic studies indicate that there
may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM (See

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities, in healthy volunteers, diltrazem has been shown to increase serum digoxin levels up to 20%

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed

Pregnancy: Category C Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality These doses, in some studies, have been reported to cause skeletal abnormatities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of still births at doses of 20 times.

the human dose or greater
There are no well-controlled studies in pregnant women, therefore use CARDIZEM in pregnant women only if the potential benefit tustifies the potential risk to the fetus

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation

Pediatric Use. Safety and effectiveness in children have not been established

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricu lar function and cardiac conduction abnormalities have usually been

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placeho therapy

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition in many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are edema (2.4%),

headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%) asthenia (1.2%), AV block (1.1%). In addition, the following eyent were reported infreguently (less than 1%) with the order of presenta tion corresponding to the relative frequency of occurrence

Flushing, arrhythmia, hypotension, bradycai Cardinyascular

dia, palpitations, congestive heart failure syncone

Nervous System Paresthesia, nervousness, somnolence tremor, insomnia, haflucinations, and amnesia Constipation, dyspepsia, diarrhea, vomiting Gastrointestinat

mild elevations of alkaline phosphatase, SGO SGPT, and LDH Dermatologic Pruritus, petechiae, urticaria, photosensitivit

Polyuria, nocturia

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes c vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-m dose of CARDIZEM

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM erythema multiforme, let kopenia, and extreme elevations of alkaline phosphatase, SGO SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral dilitazem has been limitel Single oral doses of 300 mg of CARDIZEM have been well tolerate by healthy volunteers. In the event of overdosage or exaggerate response, appropriate supportive measures should be employed i addition to gastric lavage. The following measures may be considered.

Administer atropine (0.60 to 1.0 mg). If their 8radycardia is no response to vagal blockade, administr

isoproterenol cautiously Treat as for bradycardia above. Fixed high High-Degree AV degree AV block should be treated with ca

diac pacing Cardiac Failure Administer inotropic agents (isoprotereno dopamine, or dobutamine) and diuretics. Hypotension

Vasopressors (eg, dopamine or levartereni bitartrate).

Actual treatment and dosage should depend on the severity of th clinical situation and the judgment and experience of the treatin nhysician

s in mice and rats range from 415 to 740 mg/k and from 560 to 810 mg/kg, respectively The intravenous ED_{co} 's these species were 60 and 38 mg/kg, respectively The oral ED_{co} 's these species were 60 and 38 mg/kg, respectively The oral ED_{co} 's dogs is considered to be in excess of 50 mg/kg, while lethality we seen in monkeys at 360 mg/kg. The toxic dose in man is not know but blood levels in excess of 800 ng/ml have not been associate with toxicity.

DOSAGE AND ADMINISTRATION

8lnck

Exertional Angina Pectoris Due to Atheroscierotic Com nary Artery Disease or Angina Pectoris at Rest Due to Cort nary Artery Spasm. Dosage must be adjusted to each patient needs. Starting with 30 mg four times daily, before meals and a bedtime, dosage should be increased gradually (given in divide doses three or four times daily) at one- to two-day intervals uni optimum response is obtained Although individual patients may appears to be 180 to 240 mg/day There are no available data concering dosage requirements in patients with impaired renal or hepat function if the drug must be used in such patients, titration should be carried out with particular caution

Concomitant Use With Other Antianginal Agents:

1. Sublingual NTG may be taken as required to abort acutanginal attacks during CARDIZEM therapy

2. Prophylactic Nitrate Therapy—CARDIZEM may be safe coadministered with short—and long-acting nitrates, but the have been no controlled studies to evaluate the antiangin

effectiveness of this combination

Beta-blockers. (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NC 0088-1771-49) Each green tablet is engraved with MARION on or side and 1771 engraved on the other CARDIZEM 60-mg score tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Ur Dose Identification Paks of 100 (NDC 0088-1772-47). Each yellotablet is engraved with MARION on one side and 1772 on the other last of 1772 and the other last of 1772 on the 1772 on the other last of 1772 on the 1772 on t

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CANCER CORNER

WILLIAM M. DUGAN, JR., M.D. Clinical Oncology Center, Methodist Hospital of Indiana

4th National Seminar on Community Cancer Care: The 4th National Seminar on Community Cancer Care, Oct. 17-20, 1985, will be held at the Hyatt Regency, Indianapolis.

This seminar is co-sponsored by Community Hospital (Indianapolis), Deaconess Hospital (Evansville), Methodist Hospitals (Gary), Methodist Hospital of Indiana, Inc. (Indianapolis), Parkview Memorial Regional Oncology Center (Fort Wayne), St. Francis Hospital (Beech Grove), St. Joseph Medical Center (South Bend), St. Vincent Hospital and Health Care Center (Indianapolis), Memorial Hospital (South Bend), Wabash County Hospital (Wabash), the Comprehensive Cancer Center, Ohio State University (Columbus, Ohio), Association of Community Cancer Centers (ACCC) (Washington, D.C.), and the American Cancer Society (Indianapolis).

Any person with suspected or diagnosed cancer deserves the most current and appropriate treatment available. Since 85% of cancer care is administered at the community level, organized community cancer program efforts are imperative to ensure quality patient care. The goal of this seminar is to provide information and direction about how to plan, develop, and implement components of community hospital cancer programs.

At the completion of this seminar, the participant will be able to: 1) discuss economics and ethical issues/ questions/concerns that affect community hospital cancer programs, 2) discuss possible solutions to address these issues/questions/concerns, 3) continue to identify advances in community cancer program development, and 4) evaluate the information obtained for making choices regarding individual program development for the future.

The following topics will be included in the seminar schedule:

Thursday, Oct. 17; Special Interest Day:

- Oncology Nursing Conference, 7 a.m.-4:30 p.m.
- Program Administrators' Conference, 8 a.m.-4 p.m.
- Midwest ELM Users Group, 1 p.m.-5 p.m.

Friday, Oct. 18:

Ethical Issues in Cancer Care, Economic Issues in Cancer Care, Tertiary Perspectives of Outreach Programs, Reducing Mortality: Is It Possible?, and Human Side of Humanomics. A panel discussion will give responses to Ethical Issues in Cancer Care and Economic Issues in Cancer Care.

Friday evening, 7-9 p.m., will be a special education evening at the Cancer Care Fleamarket, which will include exhibits and lots of fun for everyone.

Saturday, Oct. 19:

Clinical Research: Practical? Ethical? Advantageous? and Life After Data. Mini Sessions include: Volunteers: Helping Hands, Loving Hearts; Whose Illness is This Anyway?; and Outreach Programs. Sunday, Oct. 20:

Co-Ventures—Problems, Profits, and Promises; and Marketing: Striking the Balance Between Winning and Losing.

Entertainment for the seminar will be provided by the Indianapolis Pops Orchestra.

For further information, write Office of Continuing Medical Education, Methodist Hospital of Indiana, Inc., 1604 N. Capitol Ave., Indianapolis 46202.

CAN CANCER BE PREVENTED? Yes! Some cancers can be prevented. Eighty-five per cent of lung cancers are caused by cigarette smoking and most skin cancers by frequent over exposure to direct sunlight. However, about 160,000 people with cancer who might have been saved by earlier diagnosis and prompt treatment will die in 1985. What can you do to help prevent cancer?

- Don't smoke or use tobacco in any form.
- If you drink alcoholic beverages, do so in moderation.
 - Eat foods low in fat.
- Include fresh fruits, vegetables, whole grain cereals in your daily diet.
 - Avoid unnecessary x-rays.
- Keep yourself safe on the job by using protective devices indicated by your job description and/or supervisor.
- Avoid too much sunlight, wear protective clothing and use a sunscreen.

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PUBLIC HEALTH NOTES

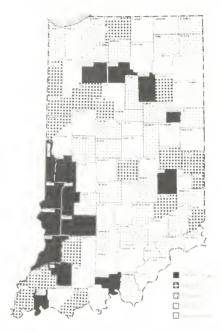
The Clean Air Act requires states to develop regulations for toxic air pollutants that contribute to "... air pollution which may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible illness."

The Indiana State Board of Health's Air Pollution Control Division is working on a plan to identify and address the sources of toxic air pollutants within the state. An initial study has been performed to determine a possible relationship between airborne toxic (noncriteria) pollutants and the cancer mortality rate (CMR). Noncriteria pollutant emissions include chemical substances that present either a shortor long-term risk to human health and that are not currently regulated by any air pollution control standard.

Data included in Indiana Vital Statistics-1982, compiled by the ISBH's Division of Public Health Statistics, were used by the ISBH to develop age-adjusted cancer mortality rates for each county and city or town in Indiana for different types of cancer. The data facilitates a division of counties and towns into five zones: more than 230, 200-230, 170-200, 140-170, and less than 140 deaths per 100,000 population (see map).

The average cancer mortality rate (ACMR) for Indiana is 185 deaths per 100,000 population. The average of the 15 maximum ACMR areas is 246 deaths per 100,000 population, approximately twice as great as the rate in the 12 minimum rural areas. The ACMR for the U.S. population as a whole was approximately 187 deaths per 100,000 population in 1982. Such a disparity indicates that a combination of factors exist within the areas, causing an increase in the incidence of cancer. The problem of establishing a correlation between airborne toxic pollutants and increased cancer mortality rates is complicated by the variety of cancer substances and the large number and diversity of sources.

However, in spite of these complications and the difficulties encountered in drawing a correlation based upon the few existing laboratory and epidemiological investigations, there is some evidence that airborne toxicants contribute to an increased rate of cancerrelated mortality. Although no strong association has been established bet-



Indiana map depicts the annual cancer mortality rate per 100,000 population (1982 figures).

ween ambient air levels of any specific compound or compounds and human cancer-related mortality, it can be presumed that the presence of carcinogens in the atmosphere of highly polluted counties contributes to the high cancer mortality rates in those areas, especially to respiratory cancer mortality rates.

Cancer of the respiratory system (51 deaths per 100,000 population) and of the digestive organs and peritoneum (47 deaths per 100,000 population) represent the highest cause of cancerrelated mortality in the state. Cancer of the lymphatic and hematopoietic tissues accounts for 17 deaths per

100,000 population, cancer of the genito-urinary organs results in 14 deaths per 100,000 population, and cancer of the breast and other reproductive organs—characteristic of females—causes 28 deaths per 100,000 population.

The level of pollution, age, and gender have an impact on cancer mortality rates. Heavily industrialized and highly polluted rural areas have much greater cancer mortality rates than rural, less polluted areas. Additionally, analysis indicates a difference in the cancer-related mortality rates of males and females. This difference is greatest in mortality rates associated with cancer of the respiratory system (37 deaths per 100,000 population for males vs. 14 deaths per 100,000 population for females). The ACMR values for cancer of the digestive organs and peritoneum are practically the same for both sexes. These observations indicate an occupational effect added to an air pollution effect.

This conclusion is supported by studies of highly polluted counties in comparison with counties with few pollutants, including cities within these counties. Studies of Vanderburgh County and Evansville, along with Allen County and Fort Wayne, indicate higher county-wide cancer mortality rates for males. However, in both cities it was found that the cancer mortality rates of females could exceed those specified for males.

Age of the individual is also an important factor in cancer-related mortality. The period between 65 and 75 years of age is critical for the majority of cancer diseases: of lymphatic and hematopoietic tissues, of breast and of reproductive organs. The critical age for cancer of the digestive organs and peritoneum, and of other genitourinary organs, is between 75 and 85 years of age.

For more information about the air toxics program, contact the ISBH Division of Air Pollution Control, 317/633-0600.

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Courting Disaster

Recollections of an Indiana Judge about Dr. E. Rogers Smith

PAUL H. BUCHANAN, JR. Indianapolis

n 1939 a first degree murder trial took place in the old Marion County Court House. Earl C. Townsend, Jr. of Indianapolis plead the Defendant not guilty by reason of temporary insanity. It seems the Defendant shot his wife five times and accompanied the act with the tender statement, "Die, you little bitch."

The defense featured Dr. E. Rogers Smith, an Indianapolis psychiatrist who gained fame as a formidable professional witness. Dr. Smith testified that the Defendant was suffering from Dementia Praecox and Paranoia and was not responsible for his misdeed due to temporary insanity.

Undaunted, the deputy prosecutor undertook a bold course of cross-examination:

Dep. Pros: Dr. Smith, were you born in Indiana?

Dr. Smith: Yes sir. Dep. Pros: Where?

The author is Chief Judge, Indiana Court of Appeals; he was previously in private practice in Indianapolis.

Reprinted with permission from Res Gestae, April 1985, Res Gestae is the official publication of the Indiana State Bar Association.

Editor's Note: Dr. E. Rogers Smith was born and raised at the Eastern Indiana Hospital for the Insane at Richmond in 1891, where his father, Dr. Sam Smith, was superintendent. He was Emeritus Professor of Neurology, Indiana University School of Medicine, at the time of his death at the age of 80, July 2, 1972.

Dr. Smith: About 5 miles east of Richmond.

Dep. Pros: Then you were just barely born in Indiana?

Dr. Smith: If you will admit that you are just a deputy prosecutor, I'll admit that I almost was born in Ohio ... laughter from jury ... gavel by Judge Dewey E. Meyers.

Dep. Pros: Dr. Smith, you have been paid to testify here today?

Dr. Smith: No, but I'd better be. Laughter from jury, gavel by Judge Meyers.

Dep. Pros: Dr. Smith, you are making some claim to this jury that you are an expert on this matter of insanity.

Dr. Smith: Yes sir, I do.

Dep. Pros: What are your qualifications?

Dr. Smith: Well, in the first place, I was born in a hospital for the insane!

Dep. Pros: Is your contention that being born in a hospital for the insane makes you an expert on insanity?

Dr. Smith: No, but do you know anyone else who had such an early opportunity to begin his studies?

Dep. Pros: Doctor, how did you happen to get born in an insane hospital? I'm sure the jury would like to hear about any members of your family who were having mental difficulties.

Dr. Smith: Even at the time of my birth, I was not capable of choosing where I would be born. My family had the usual 'mental difficulties'.

Dep. Pros: Dr., I am not pleased by your constant beating around the bush ... just answer my questions.

Dr. Smith: Well, my father was Superintendent of the Indiana State Hospital for the Insane at Richmond and my mother told me it was *forum*

conveniens as you lawyers say.

Redirect Examination:

Mr. Townsend: Dr. Smith, you are licensed to practice medicine in the State of Indiana?

Dr. Smith: I have been so licensed since 1935.

Mr. Townsend: Dr. Smith, please summarize your medical educational background.

Dr. Smith: I graduated from the University of Michigan Medical School with a major in psychiatry; I interned at Johns Hopkins Medical University at Baltimore where I received an honorary degree in psychiatry; I took the examination and received Board certification in psychiatry.

Recross Examination:

Dep. Pros: Well, Dr. Smith, with credentials such as you have I wonder if you could give this jury your opinion as to whether you are sane or insane.

Dr. Smith: Hell, I have an opinion that in general I am about as insane as you are. In cases such as this it helps.

Laughter from jury, gavel by Judge Meyers.

Re-direct:

Mr. Townsend: Dr. Smith, did you examine the defendant?

Dr. Smith: Yes sir, in his cell at the county jail the defendant often warned me to duck a flock of large white birds that kept circling ever nearer to me and diving at me. He also warned that President Roosevelt was extremely dangerous and was planning to destroy the nation by poison gases.

After three hours the jury returned a verdict of not guilty by reason of insanity.

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To obtain Category 1 credit for this month's article, complete the quiz on page 719.



Alcoholism Research: Recent Developments

ROSS McHENRY, M.D.¹ DAVID W. CRABB, M.D.² Indianapolis

"In moderation, wine, beer, and spirits may be taken throughout a long life without impairing the general health.... Chronic alcoholism is a condition very difficult to treat, and once fully established the habit is rarely abandoned. The most obstinate cases are those with marked hereditary tendency."

 $Osler\ 1896$

LCOHOLISM is a very common, very costly health problem. It contributes to enormous economic losses (an estimated \$26 billion in lost productivity and \$17 billion in health care), social disruption and loss of life in alcohol-related automobile accidents (16,000-30,000 deaths/year), alcoholic cirrhosis (over 29,000 deaths/year) and a six- to 15-fold increased rate of suicide among alcoholics.

As many as 7% of the adult American population drink to excess. It is inevitable, then, that physicians will encounter patients suffering the medical and social consequences of alcoholism. We will review the metabolism of alcohol, definitions and natural history of alcoholism, and the role of heredity in its development. We will then discuss some simple clinical tools for detecting alcoholism, and the therapy available for alcoholic patients,

including recent evidence that early diagnosis of alcoholism improves the results of therapy.

Alcohol Metabolism

Ethyl alcohol is produced in the gastrointestinal tract in small quantities and is metabolized in the liver by two major alcohol metabolizing enzymes, alcohol dehydrogenase (ADH) and a microsomal ethanol oxidizing system (MEOS). The majority of ingested alcohol is oxidized by ADH. Human ADH exists in multiple molecular forms, or isoenzymes. The pattern of isoenzymes is genetically determined by three ADH gene loci. Systematic studies of human ADH by Li and Bosron at Indiana University School of Medicine have shown that these isoenzymes differ widely in kinetic properties, and may account for the two- to threefold variation in individual alcohol elimination rates.

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MEOS increases in activity during chronic ethanol ingestion, and may play a role in the accelerated rate of alcohol metabolism observed in alcoholies.

The product of ADH and MEOS, acetaldehyde, is oxidized in liver aldehvde mitochondria bу dehydrogenase to acetate, which is subsequently oxidized in extrahepatic tissues. Aldehyde dehydrogenase has gained attention because it is inhibited by disulfiram (Antabuse), and because one form of aldehyde dehydrogenase is genetically absent in about 50% of Orientals. In patients lacking aldehyde dehydrogenase or taking Antabuse, in gestion of alcohol results in high plasma levels of acetaldehyde and a syndrome of cutaneous flushing, vasodilation, headache, nausea and hypotension. These adverse reactions are the basis for the use of Antabuse as a form of deterrent treatment of alcoholism.

Definition and Natural History of Alcoholism

Alcoholism may be defined as a persistent, progressive pattern of alcoholseeking behavior, which results in loss of control of drinking and the development of social, occupational and physical impairment. Alcoholics usually display tolerance to increasing alcohol ingestion and manifest withdrawal symptoms (e.g., tremulousness, malaise), which are relieved by drinking.

There is no single natural history of alcoholism. The social impairment of alcoholics includes work-related problems (absenteeism), family disruption (divorce and child abuse) and legal difficulties (arrests for drunken driving). Accidental injury is much more common in alcoholics than in non-drinkers. Alcoholics typically are aware that their drinking may be responsible for these problems and that attempts to control their drinking are unsuccessful.

There are consistent biological responses to chronic alcohol consumption. The microsomal drug and ethanol oxidizing enzymes (MEOS) are induced.

There is also the development of metabolic tolerance to ethanol, i.e., the ability to metabolize alcohol at a faster rate. Independently, the patient develops central nervous system tolerance, demonstrating less response to a dose of ethanol. These phenomena are difficult to assess clinically, although a patient with a blood alcohol concentration of greater than 150 mg/dl who does not appear intoxicated is probably tolerant to alcohol.

High density lipoprotein-cholesterol and VLDL-triglyceride levels are increased by low to moderate alcohol intake (four to six drinks/day). The increased rate of VLDL synthesis results from inhibition of fatty acid oxidation in the liver, and is accompanied by development of fatty liver. Alcohol ingestion, usually in excess of six drinks/day, increases the serum level of gamma-glutamyl transpeptidase (GGT), and causes macrocytosis. The macrocytosis is usually associated with normal bone marrow histology, and vitamin B_{12} and folate levels.

Two important medical complications of alcoholism are cirrhosis and chronic pancreatitis. These typically develop after five to 10 years of alcohol consumption in excess of the equivalent of about 10 ounces of distilled spirits per day. Certain HLA antigens are found more frequently in patients with these disorders than in the general population, suggesting that the risk of their development may be in part genetically determined. It has been demonstrated that typical alcoholic cirrhosis can develop in baboons fed alcohol as part of a nutritious diet, indicating that alcohol itself is a hepatotoxin. Alcoholic liver disease includes fatty liver (which in general is not a precursor of more severe liver disease), alcoholic hepatitis and cirrhosis. Only a minority (probably fewer than 30%) of alcoholics ever develop aleoholic hepatitis or cirrhosis. Hepatomegaly is common in all forms of alcohol-related liver disease. The differentiation of these forms of liver disease from each other and from other types of liver disease cannot be confidently made without a liver biopsy. This was demonstrated by a recent report which indicated that 10 to 20% of alcoholic patients with abnormal liver tests in fact have a variety of non-alcoholic liver diseases when they undergo liver biopsy. The prognosis of alcoholic cirrhosis has been shown to improve with abstinence.

Other alcohol-related medical problems are the various withdrawal syndromes (tremulousness, hallucinosis, and delerium tremens), cardiomyopathy, peripheral polyneuropathy and hypertension. Alcoholics become less visible to society as they age. Unlike younger alcoholics, older alcoholics may no longer work or drive, and may live alone, so that the destructive effect of alcoholism in these social spheres is less apparent. Like depression, alcoholism is probably more common in the elderly than has been appreciated.

Genetics and Alcoholism

There has long been a suspicion that the risk of alcoholism is to some extent genetically determined. Research in this area has focused on three areas: 1) selective breeding of animals which exhibit disparate behavioral and physiological responses to alcohol, 2) studies on inter-individual variations in response to alcohol and rates of alcohol metabolism, and 3) twin and adoptee studies of the risk of development of alcoholism in the children of alcoholics.

Animal studies have shown that it is possible to selectively breed animals which differ in the amount of alcohol taken orally in a free-choice situation, time required to recover from alcoholinduced sleep, the alcohol metabolism rate, development of tolerance to ethanol, and the severity of withdrawal symptoms after stopping ethanol consumption. This research approach has pointed out that many aspects of alcohol-related behavior can be influenced by purely genetic factors.

The development of alcohol-prefer-

ring (P) and nonpreferring (NP) lines of rats through selective breeding is most important and interesting. Studies of these animals by Li. Lumeng, Waller and McBride at Indiana University School of Medicine have shown that the P line satisfies all the requirements for an animal model of alcoholism: 1) The P rats drink 10% ethanol solutions when food and water are available, producing "pharmacologically significant" (70 to 200 mg%) blood alcohol concentrations. 2) The P rats will work to obtain ethanol when food and water are freely available. 3) As a result of chronic free-choice drinking, the P rats develop metabolic tolerance and physical dependence, including a withdrawal syndrome.

There are several differences between P and NP animals with regard to responses to ethanol. The P rats are less "sensitive" to the effects of high dose ethanol than the NP rats as determined by performance in a jumping test. They also develop tolerance more rapidly than the NP rats. The P, but not the NP, rats will self-administer ethanol by the intragastric route in a free-choice situation. This important finding clearly indicates that ethanol is a positive reinforcer and this effect is not mediated by the smell or taste of ethanol. Consistent with the positive reinforcing effect of ethanol, the P, but not the NP, rats exhibit an excitatory response to low dose ethanol as measured by spontaneous motor activity. In neurochemical studies it was found that the levels of serotonin and 5-hydroxy-indoleacetic acid in several brain areas are lower in the P than in the NP rats.

The last finding suggests that the serotoninergic neuronal pathway is an important component of the ethanol reward system. Indeed, when fluoxetine (a serotonin uptake inhibitor) was administered to P rats, intragastric self-administration of ethanol was curtailed, but the self-infusion of water and ingestion of solid food were not. The P and NP lines of rats should constitute an important animal model to

TABLE

Screening Questionnaires The Brief MAST

- 1. Do you feel you are a normal drinker? Yes (0) No (2)
- 2. Do friends or relatives think you are a normal drinker? Yes (0) No (2)
- 3. Have you ever attended a meeting of Alcoholics Anonymous (AA)? Yes (5) No (0)
- 4. Have you ever lost friends or girlfriends/boyfriends because of drinking? Yes (2) No (0)
- 5. Have you ever gotten into trouble at work because of drinking? Yes (5) No (0)
- 6. Have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking? Yes (2) No (0)
- 7. Have you ever had delirium tremens (DTs), severe shaking, heard voices or seen things that weren't there after heavy drinking? Yes (2) No. (0)
- 8. Have you ever gone to anyone for help about your drinking? Yes (5) No (0)
- 9. Have you ever been in a hospital because of drinking? Yes (5) No (0)
- 10. Have you ever been arrested for drunk driving or driving after drinking? Yes (2) No (0)

The CAGE

- 1. Have you ever felt you should CUT down on your drinking?
- 2. Have people ANNOYED you by criticizing your drinking?
- 3. Have you ever felt bad or GUILTY about your drinking?
- 4. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hang-over (EYE-OPENER).

Trauma Questionnaire

Since your eighteenth birthday:

- 1. Have you had any FRACTURE or dislocations to your bones or joints?
- 2. Have you been injured in an ASSAULT or fight (excluding sports)?
- 3. Have you been INJURED after drinking?
- 4. Have you been injured in a road TRAFFIC accident?
- 5. Have you injured your HEAD?

study the neurochemical and neurophysiological mechanism(s) responsible for the alcohol reward system. It should also be a useful test system to investigate the factors and pharmacologic agents which can enhance or decrease free-choice alcohol ingestion.

Several biological responses of humans to alcohol have been found to vary between individuals. Much of this variation appears to be genetically determined. For instance, the electroencephalographic (EEG) response to alcohol ingestion varies between individuals, but is nearly identical in identical twins. The rate of alcohol elimination varies by two- to threefold between individuals; twin studies have shown that about half of this variation may be due to genetic factors. The inheritance of different combinations of ADH isoenzymes could account for this variation, but it has not yet been possible to determine the isoenzyme pattern

of human volunteers in order to correlate it with the rate of alcohol metabolism.

More direct evidence for a genetic basis for alcoholism has been obtained from family studies. Alcoholics have a higher probability of having alcoholic relatives than non-alcoholic controls. However, families share both genetic characteristics and environments. Studies on the adopted-out children of alcoholics have provided important information regarding the relative importance of environmental and genetic factors in alcoholism. These studies have repeatedly demonstrated that adopted-out children of alcoholics are two to three times more likely to become alcoholics compared with adopted-out children of non-alcoholics. Also, a subgroup of adopted-out sons of alcoholic fathers has up to a ninefold increased risk of developing alcoholism, which is independent of the environment in which they were reared. In addition, children of alcoholics display abnormal results on neuropsychological testing and distinct EEG and evoked potential responses even when not administered ethanol. These recent discoveries are important for the development of alcoholism detection and prevention strategies for children of alcoholics.

Clinical Clues to the Diagnosis of Alcoholism

Physicians are told, often by nonphysicians, that they frequently misdiagnose, underdiagnose or ignore alcoholism. It is recognized that alcoholics, although concerned about their addiction, do not often approach physicians specifically for help to stop drinking. Because of the many nonspecific manifestations of alcoholism and the overlap between socially acceptable and behaviorally dangerous alcohol consumption, alcoholics commonly remain undiagnosed early in their disorder. Clearly, there is a need for simple and reliable tests to detect unsuspected alcoholism. Several questionnaires (Table) and laboratory tests

have been evaluated for this purpose.

Historical Clues: The Table describes three short questionnaires which are useful in detecting early alcohol abuse. Pokorny et al. extracted the 10 most discriminating items from the Michigan Alcoholism Screening Test (MAST), a 25-item questionnaire developed for psychological testing, to produce the Brief MAST (Table). This test requires less than five minutes to administer and correlates well with the standard MAST. They found that a cumulative score of six or greater on the brief test identified all the alcoholics and misidentified only 11% of the non-alcoholics in hospitalized psychiatric patients. Another rapid screening test is the CAGE questionnaire (Table). Two or more positive responses indicate a high likelihood of alcoholism. Mayfield et al. studied 366 psychiatric patients with a 33% prevalence of alcoholism, as determined by the MAST. The CAGE test had a true-positive rate of 81%, with a false-positive rate of 11%. Recently, Skinner et al. developed a trauma questionnaire (Table), which can be recalled by the mnemonic FAITH. Two or more positive responses were considered suggestive of alcoholism. It had a sensitivity of 70% for detecting alcoholism in family practice patients compared with the MAST.

These studies suggest that, by incorporating some of the questions used in these tests into the review of systems, or by administering these tests to patients, alcoholic patients may be diagnosed at an early stage. We suggest that the CAGE and trauma questions be asked during routine historytaking and that an estimate of frequency and amount of alcohol drunk be obtained. Suspicion of alcoholism can be confirmed with the MAST or by questioning family members.

Laboratory Tests: The only true biochemical test of alcohol consumption is the presence of alcohol or its metabolites in a patient's body fluids. A blood alcohol level of greater than 300 mg/dl at any time or over 100 mg/dl during routine examinations is an important indicator of alcoholism. A blood alcohol level greater than 150 mg/dl in a patient who is not obviously intoxicated represents tolerance and is also an indicator of alcoholism. Recent studies have found that acetaldehyde. formed during ethanol metabolism, irreversibly reacts with hemoglobin, forming a modified hemoglobin. The level of this hemoglobin might be used to monitor recent alcohol intake in a manner similar to the use of hemoglobin A_{le} in diabetics. This test is still in the development stage.

All other available laboratory tests are both rather insensitive and nonspecific for alcoholism. Because of this, their usefulness varies depending on the patient population under study. Elevations of HDL-cholesterol are present in 50-80% of alcoholics. However, the HDL-cholesterol level is reduced in patients with liver disease and, thus, is not a reliable test for detecting alcoholism in patients with liver disease. Alcoholism should be considered in patients with hypertrigly-ceridemia.

None of the commonly ordered liver enzymes, e.g., transaminases or alkaline phosphatase, are sensitive or specific indicators of excessive alcohol ingestion. However, elevated GGT levels occur in 70%-80% of alcoholic patients before other tests become abnormal, presumably due to induction of the enzyme. An elevated GGT is not specific for alcohol ingestion since it may be elevated in non-alcoholic liver disease and with ingestion of drugs known to induce liver enzymes.

The most frequent hematologic abnormality in alcoholics is a raised mean corpuscular volume (MCV). In a survey of 8,000 company employees, 3% had macrocytosis, and over 90% of these individuals admitted to heavy alcohol consumption. In another study, 89% of alcoholics had an MCV greater than 90 fl compared to 24% of patients with non-alcoholic chronic liver disease.

Computerized statistical analysis of

commonly ordered laboratory tests (SMA-20 and blood count) in patients with varying levels of alcohol consumption correctly classified 100% of nonalcoholics without liver disease, 98% of alcoholic patients with presumed mild alcoholic liver disease, 96% of alcoholics with significant liver disease and 89% of non-alcoholics with liver disease. Although few practitioners will be able to use this computerized statistical analysis, these studies indicate that attention to abnormal serum lipids, an elevated MCV or GGT level, especially in combination, may reveal unsuspected alcohol abuse. However, there remains the need for a test which is specific for alcoholism and which quantifies the amount of alcohol drunk over time.

Conventional Treatment of Alcoholic Patients

The traditional methods of treatment have usually been applied to patients with advanced alcohol abuse and physical dependence. Detoxification, the process of withdrawing the alcoholdependent patient from alcohol and controlling the withdrawal symptoms, has usually been done in the hospital. However, outpatient detoxification programs are an alternative for patients without serious alcohol dependence or underlying medical illness.

Long-term treatment of alcoholism can be aimed at either reducing the patient's craving for alcohol, or at increasing the patient's ability to resist that craving. At present, fluoxetine is being evaluated for its ability to reduce this craving. Uncontrolled studies of alcoholics treated with chlordiazepoxide (Librium) have reported a lower short-term dropout rate from alcohol treatment programs when compared with placebo, imipramine or thioridazine treatment. To our knowledge. there are no studies documenting the effectiveness of long-term benzodiazepine therapy of alcoholics, and this cannot be recommended.

The other alternative, helping the

patient to resist alcohol craving, is the goal of conventional therapy. Psychotherapy, group therapy, Alcoholics Anonymous, aversion therapy, and behavior modification techniques all rely on teaching and persuading the patient to remain abstinent. There is no convincing evidence that one form of treatment is superior to the other, and they all have relatively high relapse rates. Therefore, more coercive strategies, such as voluntary and supervised Antabuse therapy, have been developed.

Antabuse has been used in the treat ment of alcoholism for over 30 years. However, most of the early studies claiming that Antabuse is effective had significant design problems. Fuller and Roth performed a controlled doubleblinded, prospective clinical trial of Antabuse in the treatment of a group of 128 alcoholic men. The patients were assigned to one of three groups: group 1 received the usual dose of Antabuse (250 mg/day), group 2 received a pharmacologically ineffective dose of Antabuse (1 mg/day), and group 3 received a riboflavin placebo. Only the patients receiving either dose of Antabuse were warned of the risk of ingesting alcohol. At one year, the abstinence rates for the two Antabuse groups were nearly identical (21% and 25%) compared with the placebo group (12%). The patients who relapsed were found to have voluntarily discontinued Antabuse several days before returning to drinking. The authors concluded that it is the fear of the Antabuse reaction that is important in preventing drinking. Supervised Antabuse therapy, i.e., by a spouse, employer, or the courts, may be more effective than voluntary therapy.

Antabuse has several adverse side-effects. It can exacerbate depression, schizophrenia, and seizure disorders. Patients drinking while taking Antabuse have died from myocardial infarction or cerebrovascular accidents. Contraindications to Antabuse use include myocardial disease, severe pulmonary or hepatic disease, chronic

renal failure, psychosis, and severe depression. Alternatively, Fuller and Roth's findings suggest that a pharmacologically inactive dose of Antabuse is as effective as standard dose therapy and lacks the potential for adverse reactions.

Early Detection and Treatment of Alcohol Abuse

Conventional treatment of alcoholic patients is usually instituted at a relatively late stage, when the patient is often unemployed and has lost important family support. There has been a general movement toward earlier identification of alcoholics before physical dependence and medical complications occur. This has been an impetus for the evaluation of the screening tests discussed above. Recently, studies conducted in Europe have suggested that early detection and treatment is both feasible and effective.

In Sweden, middle-aged alcoholics identified by means of elevated GGT values were randomly assigned to a control group receiving advice to limit alcohol consumption, or to an intervention group. The latter group was repeatedly encouraged to drink less, and received feedback about their GGT levels. Compared to controls, the intervention group showed significant reductions in work absenteeism, hospitalization and mortality at up to six years after initial screening. In France, screening for problem drinking is routinely conducted in industry, health care settings and by the courts. using a simple clinical exam and biochemical tests (GGT and MCV). Alcoholics so identified are referred to early treatment clinics, the efficacy of which is being evaluated.

These programs demonstrate that early detection and intervention are now feasible in non-medical settings. Prospective controlled studies should determine whether early detection and intervention can alter the natural history of alcoholism.

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Resident Physician Essay Contest

During the 1985-86 resident training year the Resident Medical Society of the ISMA and INDIANA MEDICINE will conduct a medical essay contest. All members of the Resident Medical Society are eligible to enter. Each author may choose the subject. Essays (medical articles) will be limited in length to 10 pages of typescript, properly margined and double spaced. Illustrations are encouraged and are to be included in the space limitation.

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Judges will consist of two specialists within the field of the article, three medical education directors from Indiana institutions with approved residency training programs, and one member of the INDIANA MEDICINE Editorial Board.

Authors of the articles judged to be the four best will receive prizes of \$100 each, and an additional \$100 prize will be given to the best article of the four. All prize-winning articles will be published in INDIANA MEDICINE. It is hoped that space will allow the publication of other entries to be chosen in the order of their numerical scores.

Essays will be judged on the following criteria: SCIENTIFIC MERIT: Research methods, data analysis, subject or patient population selection, control of variables, literature analysis.

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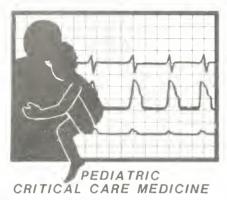
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Pediatric Epiglottitis



PIGLOTTITIS is an acute inflammation and edema of the epiglottis and aryepiglottic folds most often caused by Hemophilus influenzae type B. Characterized by abrupt onset and rapid progression to lifethreatening upper airway obstruction in hours, immediate diagnosis and management is essential. This article will discuss the diagnosis and management of pediatric epiglottitis. In the next issue of INDIANA MEDICINE, we will review the Methodist Hospital experience with over 30 pediatric epiglottitis patients.

Presentation

The child with epiglottitis is typically 2-6 years old. Fever, irritability, and sore throat followed by dysphagia, dysphonia, drooling, and inspiratory

From the Pediatric Intensive Care Unit, Methodist Hospital of Indiana, Inc.

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respiratory distress rapidly occurs over a 12-18 hour period. An anxious, toxic, febrile child with variable degrees of respiratory distress is typical.

The so-called "airway-preserving posture" at presentation is the sitting position with forward flexion at the waist, slight cervical flexion, forward chin thrust with mouth open, and tripod placement of the supporting upper extremities.4 It is crucial that the child with suspected epiglottitis be allowed to assume his desired position to avoid acute total airway obstruction. Restlessness, anxiety, and air hunger suggest hypoxemia and advanced airway obstruction. Cyanosis, exhaustion, lethargy, and deep sternal retractions suggest respiratory failure and the need for immediate oxygenation and ventilation with establishment of an artificial airway.

Clinical history and emergency room (ER) exam usually differentiate epiglottitis from a more common and usually less serious cause of infectious upper airway obstruction in children, viral laryngotracheobronchitis (LTB; viral eroup). Laryngotracheobronchitis is characterized by a more gradual onset of stridor following several days of upper respiratory tract symptoms in a child less than three years. A harsh barking cough, hoarse voice, low-grade fever, lack of toxicity, waxing and waning course over several days, and seasonal incidence suggest the diagnosis of LTB rather than epiglottitis. Drooling and dysphagia is uncommon in patients with LTB.

Emergency Room Evaluation and Management

A child with possible epiglottitis, or with marked upper airway obstruction from any cause, should be triaged to a constant care area equipped for emergency intubation and tracheostomy. A physician should immediately be notified of the patient's arrival. A quick history from parents should establish the progression, timing, and duration of symptoms. The child should be allowed to maintain the position in which he is most comfortable and should never be restrained. Movement around the child should generally be slow and deliberate. A parent should be allowed to hold the child to minimize anxietv and further airway obstruction.

Oxygen by mask should be delivered only if tolerated without increased anxiety. A parent may help by holding the oxygen mask near the patient's face. Minimally disturbing the child is a major ER priority. Blood drawing and intravenous access are postponed until an airway is being or has been established, optimally in the operating room (see below). Examination of the pharynx or epiglottis may precipitate total airway obstruction and/or vagally mediated cardiorespiratory arrest and is generally contraindicated.

Much of the initial examination can be made from a distance. Particular attention should be paid to general appearance, color, posture, airway sounds, drooling, state of consciousness, and degree of respiratory distress (retractions, use of accessory muscles, dyspnea).

The diagnosis of epiglottitis is usually apparent from the history and physical exam. A lateral neck x-ray (Figure) is generally unnecessary, especially if the procedure scares the child and if it delays airway establish-

ment. If the patient is significantly obstructed and the diagnosis is in doubt, the diagnosis and subsequent management should ideally occur in the operating room. If a lateral neck x-ray is obtained when history and signs are equivocal, a portable x-ray taken in the ER is preferable to transporting the patient to the radiology department. When the diagnosis of epiglottitis is likely, whether the child is first seen in the office or ER, a physician should accompany the patient until an airway is established and the patient is admitted to the intensive care unit (ICU).

Subsequent Management

During the late 1950s, it became apparent that mortality from epiglottitis could be markedly reduced if tracheostomy was electively performed before the onset of severe respiratory distress and respiratory arrest which may occur without gradual change or prior warning.5 The safest approach to the pediatric patient with epiglottitis, whether the airway is in immediate jeopardy or not, is tracheostomy or nasotracheal intubation performed in the operating room as soon as possible after diagnosis. Medical management and observation without an artificial airway is generally not considered an option given the rapidity with which respiratory distress and respiratory arrest can occur. A well recognized team (ER physician, surgeon, anesthesiologist, operating room and ICU personnel) following an established protocol is necessary for rapid airway placement. Practically, this is not always possible in every hospital setting. Rapid, physicianaccompanied transport to a facility where this team approach is available then becomes necessary.

The patient should be accompanied to the operating room by parent, physician, and equipment for bag and mask oxygen/ventilation and intubation. Preparation for bronchoscopy and tracheostomy is routine. Typically, the patient receives an oxygen/halothane anesthesia, and either nasotracheal in-



FIGURE: Demonstrates enlarged epiglottis (tip of arrow), edematous aryepiglottic folds, loss of vallecular air shadow, hypopharyngeal dilatation.

tubation or tracheostomy over an established airway (orotracheal tube or bronchoscope) is performed.

The decision to perform a tracheostomy or nasotracheal intubation rests with the responsible physician and his/her comfort with how the airway will be maintained in the ICU setting. An intravenous line is established and blood for lab values and culture is obtained in the operating room, along with a chest x-ray to assure optimum airway position. Cleft palate elbow restraints may be applied to prevent accidental extubation.

In the ICU, airway patency and the prevention of accidental extubation are the priorities. Sedation (Morphine 0.1 mg/kg IV, Valium 0.1 mg/kg IV) may be necessary, assuming hypoxemia is not the cause of agitation. Routine saline instillation and suctioning of the nasotracheal tube or tracheostomy is mandatory. Supplemental humidified

oxygen by T-tube should always be provided, given the common hypoxemia present with epiglottitis regardless of the relief of airway obstruction or of the presence of a normal chest x-ray. Some patients with atelectasis, pneumonia, or pulmonary edema (to be discussed in next month's article) may require positive endexpiratory pressure, mechanical ventilation, and/or aggressive chest physical therapy. Ampicillin (200 mg/kg/day IV) and chloramphenicol (100 mg/kg/day IV) are begun, pending blood culture and sensitivity results.

Nasotracheal intubation, relative to tracheostomy, generally offers the advantage of earlier extubation and a shorter hospital stay. When lower airway disease does not complicate epiglottitis, successful morning extubation at 36-48 hours is typical. Direct visualization of the epiglottis and aryepiglottic folds by nasal fiberoptic bronchoscope or sedation and direct laryngoscopy in the ICU or operating room may help confirm the clinical impression of resolution of obstruction. Most patients can be discharged by the fourth or fifth day of hospitalization to complete a minimum of 10-12 days of antibiotic therapy.

Accurate diagnosis, routine early provision of an artificial airway, and excellent ICU airway care are required for optimum management of epiglottitis, a truly life-threatening cause of upper airway obstruction in children.

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Acute Renal Failure: Contemporary Management

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The Author
Reviews Recent
Developments and
Novel Approaches
to the Patient
with Acute
Renal Failure

■HE MAJORITY OF patients who survive an episode of acute renal failure recover spontaneously within four to six weeks following the precipitating event. Treatment in its various forms is therefore designed to prevent complications during the time the kidneys are unable to perform their excretory, regulatory, and endocrine functions. If the necessary steps are taken to achieve this goal, and if the patient does not succumb to the underlving disease or an associated catastrophy, recovery of renal function can be expected. General principles of management are outlined in detail elsewhere. The purpose of this report is to briefly review more recent developments and novel approaches to the patient with acute renal failure.

Dialysis Frequency

In spite of improvements in supportive management, including nutrition, nursing care, respiratory support, management of infectious complications, and new approaches to dialysis and related care, the mortality of acute renal failure has not appreciably changed in the past three decades.2 For example, the mortality rate for soldiers sustaining acute renal failure during the Korean War (68%), was not significantly different from the 63% mortality rate reported from the Vietnam conflict.3,4 Figures from civilian populations are similar, when cases of similar complexity are compared.

It is probable that current patients with acute renal failure are more likely to be critically ill, exhibit the failure of multiple organs, and are those patients who would have succumbed to their underlying illness prior to the development or recognition of acute renal failure in previous times. Thus,

Routh *et al.*⁵ derived a clinical severity score, which considered severity of illness. Their results indicated that in spite of an increasing severity of underlying illnesses, the survival rate of patients with acute renal failure improved only slightly between 1969 and 1979.

Investigators have attempted to formulate predictor variables to enable a more reliable prognosis to be established. Clinical indicators have been unsuccessful in this regard.2 However, recently Ozawa and colleagues6 reported that biochemical data reflecting hepatic energy deficit expressed as the ratio between acetoacetic acid and beta-hydroxybutyric acid provided clinically useful prognostic information. This indicator of mitochondrial redox potential successfully separated salvageable patients from those with no hope for survival. Terminal patients with very low acetoactetate-betahydroxybutyrate ratios invariably had multiple organ failure.

Dialysis frequency is considered to influence outcome in the management of acute renal failure.7 Early studies indicated that patients who were dialyzed on a regular schedule designed to maintain BUN values below 125 mg/dl had fewer complications, particularly gastro-intestinal hemorrhage, compared to those who had less frequent treatments." Experience from the Vietnam War showed no difference in BUN values between survivors and nonsurvivors; however, the management of both groups was such that BUN values were maintained at < 100 mg/dl.

Adequacy of dialysis has been tested rigorously only in patients with chronic renal failure. In such patients higher BUN values were associated with higher morbidity. Current nutritional

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interventions mandate frequent (daily if necessary) treatments to deal with fluid management and catabolism. The consequences of a high catabolic rate coupled with intermittent dialysis treatments include central nervous system dysfunction, impaired respiratory gas exchange, and acid-base dysequilibration. Thus, the issue of dialysis frequency has assumed lesser importance. Indeed, some forms of current intervention provide for gradual, albeit continuous, waste product removal.

Continuous Arterio-Venous Hemofiltration

A recent adjunct to dialytic management of patients with acute renal failure is the use of continuous arteriovenous hemofiltration.11,12 This technique employs a hemofilter device composed of hollow fiber capillaries permeable to water and solutes in blood (Fig. 1). Thus, the filter allows removal of plasma water and its dissolved solutes from the vascular space while conserving the cellular and protein constituents of circulating blood. Since the system relies on direct access to the circulation, systemic blood pressure provides the work to achieve sufficient blood flow across the filter.

The advantages of the hemofiltration approach are readily apparent. Since the patient's blood pressure provides the driving force for the treatment, should the patient's blood pressure decrease, the filtration rate will decrease pari passu and the severity of hypotensive episodes is minimized. Support with vasopressors is possible under these circumstances. The continuous nature of the therapy effectively deals with questions of dialysis frequency, disequilibrium, catabolic rate, etc. The rate of filtrate removal (600 ml/hr) allows the administration of large amounts of fluid as necessitated by modern nutritional approaches such as total parenteral nutrition. Finally, expensive mechanical devices are not necessary,

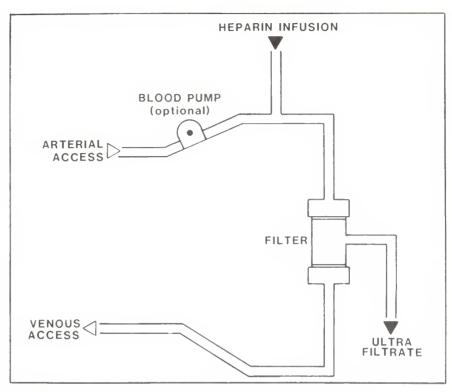


FIGURE 1

and the skill of a dialysis nurse in continuous attendance is not required.

A simple diagram of the continuous arteriovenous hemofiltration system is shown in Figure 1. Complications do occur with this technique. Hemorrhage from systemic anticoagulation appears to be the greatest risk.11,12 Anticoagulation with heparin is necessary to maintain the integrity of the filtration device. We generally administer 20 units/kg two minutes before blood flow to the filter is allowed. Familiar monitors of anticoagulation are employed. A reasonable heparin infusion rate is 500 units/hr, (corresponding to 20 ml/hr of 12,500 units per 500 ml 5% glucose in normal saline).

The clinical value of continuous arterio-venous ultrafiltration appears to be greatest in the management of patients with vascular instability. The treatment provides acceptable levels of urea nitrogen and fluid balance even in patients requiring very aggressive total parenteral nutrition. The com-

bination of these therapies may promote anabolism and a decrease in the rate of urea nitrogen production.¹²

Nutrition

The last 10 years has seen a major emphasis placed on the nutrition of the critically ill. In addition to careful fluid and electrolyte management and the administration of a regimen designed to minimize the catabolism of endogenous protein coupled with exogenous protein restriction, investigators developed the novel idea that a limited nitrogen intake given either as "high quality" protein or as a mixture of essential amino acids could reduce net urea production and improve nitrogen balance.

This approach was expanded by Lee, et al.¹³ to include intravenous administration of a solution designed to satisfy the earlier established principles. Abel, et al.¹⁴ conducted a prospective, randomized trial of parenteral nutrition compared to an equicaloric

glucose solution, and reported improved survival and more rapid recovery of renal function in patients with acute renal failure. Subsequent, additional refinements include tailored regimens, detailed information on energy requirements (see *Table 1*), and the use of special keto analogues of essential amino acids.¹⁵

Specific recommendations for therapy suggest several conclusions. First, the heterogeneous and selflimited nature of the illness mandates an eclectic approach to therapy. It is unlikely that one treatment regimen should be used for all patients with acute renal failure. Combined use of enteral and parenteral nutrition should be instituted whenever possible, even if only a fraction of total calories required and only a portion of the necessary nitrogen can be given by the enteral route. Second, despite refinements, nutritional therapy, like dialysis, has not made a significant impact on morbidity or mortality of patients whose renal insufficiency is complicated by multiple organ failure and resultant catabolism. Third, methods of controlling the catabolic response are necessary so that the beneficial effects of nutrition can be best utilized.

Infection

With the advent of modern fluid and electrolyte management and dialytic techniques, infection remains the most common cause of death in patients with acute renal failure. This conclusion has remained unchanged in the past 20 years despite the advent of ever improved antibiotic regimens and more aggressive surgical approaches to infected patients. Recent research into the treatment of gram-negative bacteremia and shock with human antiserum promises to eventually alter this unfortunate state of affairs.

Although not yet thoroughly or specifically tested in patients with acute renal failure, the results of a prospective, randomized, double blind trial of septic patients with human antiserum to mutant Escherichia coli sug-

TABLE 1
Calculation of Energy Requirements
Energy requirements in keal/day =
Basal Metabolic Requirements x 1.25 x Stress Factor

a. Basal Metabolic Requirements Weight (kg) 50 55 60 65 70 75 80 BMR (kcal/day) 1316 1411 1509 1602 1694 1784 1872 b. Stress Factor Early starvation 0.85-1.00 Burns Postop (no complications) 1.00-1.05 10-30% Surface Area 1.50 Long Bone Fracture 1.15-1.30 30-50% Surface Area 1.75 Cancer 1.10-1.45 >50% Surface Area 2.00 Peritonitis 1.05-1.25 Severe infection/ multiple trauma 1.30-1.55			4						
BMR (kcal/day) 1316 1411 1509 1602 1694 1784 1872 b. Stress Factor Early starvation Postop (no complications) 0.85-1.00 Burns Postop (no complications) 1.00-1.05 10-30% Surface Area 1.50 Long Bone Fracture 1.15-1.30 30-50% Surface Area 1.75 Cancer 1.10-1.45 >50% Surface Area 2.00 Peritonitis 1.05-1.25 Severe infection/	a. Basal Metabolic Requirements								
b. Stress Factor Early starvation	Weight (kg)	50	55	60	65	70	75	80	
Early starvation 0.85-1.00 Burns Postop (no complications) 1.00-1.05 10-30% Surface Area 1.50 Long Bone Fracture 1.15-1.30 30-50% Surface Area 1.75 Cancer 1.10-1.45 >50% Surface Area 2.00 Peritonitis 1.05-1.25 Severe infection/	BMR (keal/day)	1316	1411	1509	1602	1694	1784	1872	
Postop (no complications) 1.00-1.05 10-30% Surface Area 1.50 Long Bone Fracture 1.15-1.30 30-50% Surface Area 1.75 Cancer 1.10-1.45 >50% Surface Area 2.00 Peritonitis 1.05-1.25 Severe infection/ 1.05-1.25	b. Stress Factor								
Long Bone Fracture 1.15-1.30 30-50% Surface Area 1.75 Cancer 1.10-1.45 >50% Surface Area 2.00 Peritonitis 1.05-1.25 Severe infection/	Early starvation		0.85-1.00		Burns				
Cancer 1.10-1.45 >50% Surface Area 2.00 Peritonitis 1.05-1.25 Severe infection/	Postop (no complications)		1.00-1.05		10-30% Surface Area 1.50				
Peritonitis 1.05-1.25 Severe infection/	Long Bone Fracture		1.1	5-1.30	30-50% Surface Area 1.75				
Severe infection/	Cancer		1.10 - 1.45		>50% Surface Area 2.00				
	Peritonitis		1.05 - 1.25						
multiple trauma 1.30-1.55	Severe infection/								
	multiple trauma		1.3	0-1.55					

gest that such approaches will increase the survival of patients with gramnegative septic shock. Ziegler, et al.16 treated 103 patients suffering from endotoxic shock with human antiserum to endotoxin (lipopolysaccharide) core. A total of 109 patients received sera from the same donors prior to the donor's immunization. The number of deaths in the bacteremic patients was 42 of 109 (39%) in controls and 23 of 103 (22%) in recipients of Escherichia coli antiserum (p < 0.02). This significant difference persisted in those with profound shock. The authors concluded that it would be practical to administer human antitoxin if protective immunoglobulin could be obtained in a form suitable for regular intravenous

These promising results indicate that new modes of therapy directed against the most serious and common forms of infection in acute renal failure patients will be forthcoming in the near future.

Preventive Strategies

Vigilence equals avoidance! This caveat applies particularly to the administration of potential nephrotoxins, aminoacid containing solutions, avoidance of dehydration, concomitant fluid and electrolyte disorders, acidosis, hypoxia, etc. However, more

specific, potentially useful possibilities to minimize the chances for the development of acute renal failure are in stages of development. In addition to the well recognized maneuvers of adequate hydration and volume expansion, particular pharmacological interventions show promise. Brezis, et al.17 emphasized the vulnerability of the thick ascending portion of Henle's loop because of its particularly large energy and oxygen demands. They found that polyene antibiotics such as amphotericin B and nystatin increased membrane permeability and thus increased the amount of oxygen consumed. Extensive damage could be prevented if reabsorptive transport was inhibited by ouabain.

Similarly, aminophylline, which inhibits adenosine receptors, can protect from amphotericin B toxicity, presumably by decreasing the activity of tubuloglomerular feedback.18 Several reports indicate that drugs that inhibit calcium entry into cells protect from both nephrotoxic and ischemic acute renal failure. The mechanisms may involve an alteration of mitochondrial ealcium accumulation which in turn appears to contribute to cell injury and death.19 These lines of research raise the strong hope that preventive pharmacological intervention may be available in the not too distant future.

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Blunt Chest Trauma

An Assessment of Chest X-Ray Findings in the Diagnosis and Exclusion of Aortic Rupture

Abstract

The chest x-rays of 86 consecutive patients with blunt chest trauma and suspected aortic rupture were reviewed. Thirteen patients had surgically confirmed aortic rupture and 73 had no evidence of rupture on aortography or surgical exploration. Sixteen radiographic signs were analyzed separately and in combination both for the positive diagnosis and the exclusion of aortic rupture. No single sign nor combination of signs could reliably predict the positive diagnosis of aortic rupture in all patients with aortic rupture. No single sign could reliably exclude aortic rupture in those patients without aortic rupture. However, a combination of four chest x-ray findings can be used to exclude aortic rupture in blunt chest trauma. If the aortic knob and contour were normal and there was no deviation of the nasogastric tube or trachea, there was no case of aortic rupture in four years of experience.

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NLY 10-20% of patients with blunt chest trauma and aortic rupture survive long enough to reach the emergency room. Of the patients reaching the hospital, 25% will die within 24 hours and up to 90% within four weeks.^{2,3} Clinical findings in aortic rupture are often not specific in diagnosing or excluding aortic rupture and are reported accurately in only 45% of confirmed cases.46 Several clinical reports advocate arteriography for all patients with blunt chest trauma.7,8 Multiple radiographic findings have been reported to be associated with aortic rupture, none of which are totally reliable.

Few reports have focused on the exclusion of aortic rupture based on chest x-ray findings. Our study was twofold:

1) to assess plain film criteria individually and in combination for the positive diagnosis of aortic rupture, and 2) to look for plain film findings individually and in combination for the reliable exclusion of aortic rupture.

Methods

All patients who presented to the Emergency Room Section of the Methodist Hospital of Indiana, Indianapolis, with blunt chest trauma and who had aortography or surgical ex-

ploration for suspected aortic rupture were included in this study. Patient selection for arteriography was based on clinical grounds as determined by the Emergency Department physicians and attending surgeons and represents four consecutive years of experience. Sixteen separate plain chest film findings were analyzed separately and in combination using Fischer's exact test for their significance in the diagnosis or exclusion of aortic rupture without knowledge of the arteriographic or surgical findings. The arteriograms, surgical and clinical records were subsequently reviewed.

All chest x-rays were anteroposterior (AP) supine with a 100 cm focalfilm distance using our standard ER portable techniques. The 16 chest film findings are enumerated in the Table. Nasogastric (NG) tube and tracheal displacement were considered positive if the left lateral wall of the NG tube or trachea was positioned to the right of the T4 spinous process. Eight cm was considered the maximum normal width of the mediastinum. The aortic knob and descending thoracic aortic contour were evaluated separately. The left mainstem bronchus was judged depressed if it was greater than 40° below the horizontal. Widening of the left paravertebral stripe was considered positive if the width of the stripe exceeded one-half the width of the descending thoracic aorta. The mediastinal width-chest width (M/C) ratio was calculated by dividing the width of the mediastinum at the level of the aortic arch by the chest width at that same level.

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Results

Six signs approached statistical significance when used separately including nasogastric tube displacement, loss of aortic contour, loss of aortic knob, tracheal displacement, depressed left mainstem bronchus and left apical cap. As can be seen from the Table, each of these six signs had both false-negatives and false-positives. Each sign evaluated had false-negatives and positives, making the use of any individual sign unreliable. No combination of signs could be found to reliably diagnose all cases positive for aortic rupture. Four signs were found to be useful to exclude aortic rupture. Normal visualization of the aortic knob and contour and lack of deviation of the nasogastric tube and trachea excluded aortic rupture in four consecutive years of experience.

Discussion

Aortic rupture in blunt chest trauma is highly lethal and requires early recognition for prompt surgical repair. Since neither history nor physical findings are sufficiently accurate for the diagnosis of aortic rupture, other means, specifically chest x-rays, have been used to detect aortic rupture. Previous reports have suggested various findings to be associated with aortic rupture. 914 Other authors point out the lack of total reliability of these signs, with both false-positives and false-negatives occurring. 1519

One of the most well known and widely used signs is mediastinal widening. All patients in this study had chest films interpreted by the attending physician as subjectively demonstrating a wide mediastinum. Using the objective measurement of 8 cm, 59 of 86 patients had abnormal mediastinal widths. There was not a statistically significant difference between those with a ortic rupture and those without rupture. Likewise, there was not a significant difference in the mediastinal-chest width ratios in the two patient groups. We have not found mediastinal widening, either subjective interpretation or objective measurements, to be helpful in distinguishing patients with aortic rupture from those without rupture.¹⁹

Our study confirmed the fact that no single sign could be used to diagnose or exclude aortic rupture in every patient. Furthermore, no combination of signs was found that could reliably diagnose all cases of aortic rupture. We found, however, that the combination of normal visualization of the aortic

knob and contour and no deviation of the nasogastric tube or trachea was reliable in excluding aortic rupture. We had a 0% incidence of aortic rupture using these four signs in 86 consecutive patients representing four years of experience at a major trauma center for Indianapolis and surrounding areas.

On the basis of this study we suggest the following approach to the patient with blunt chest trauma. Patients who are critically injured require in

	TOTALS										
		+ RUPTURE 13 TOTAL			RUPTURE 73 TOTAL						
	p	+	-	?	NS	NP	+		?	NS	NP
1. NASOGASTRIC TUBE DISPLACEMENT	.0002	5	2	1		5	3	49			21
2. WIDE MEDIASTINUM	. 15	11	2				48	25			
3. LOSSAORTIC CONTOUR	. 04	12	1				47	26			
4. LOSSAORTIC KNOB	. 06	11	2				42	31			
5. TRACHEAL DISPLACEMENT	.01	5	6	1	1		8	64	1		
6. DEPRESSED LEFT MAIN BRONCHUS	. 0025	5	7		1		4	67	1	1	
7. HEMOTHORAX RIGHT	. 17	0	13				10	61	2		
8. HEMOTHORAX RIGHT	. 34	5	8				21	52			
9. PNEUMOTHORAX RIGHT	. 35	2	11				6	66	1		
10. PNEUMOTHORAX LEFT	.23	2	11				4	68	1		
11. LUNG CONTUSION	. 23	5	8				17	53	3		
12. RIB FX RIGHT	. 28	1	12				14	58	1		
13. RIB FX LEFT	. 20	6	7				22	51			
14. LEFT APICAL CAP	.01	8	5				18	53	1	1	
15. WIDE LEFT PARASTRIPE	. 13	2	3		8		4	35		34	
16. M/C RATIO	.63	Meai	n ·	39	SD =	. 05	Me	an -	. 37	SD	.09

Questionable

NS Not Seen

NP Nasogastric Tube Not Present

n n value

itial stablization, frequently with a central venous line, an endotracheal tube and often a nasogastric tube to decompress the stomach. A chest film is then obtained following the initial stablization and nasogastric tube insertion. If the nasogastric tube and trachea are not deviated and the aortic knob and contour are normally visualized, aortic rupture is unlikely (no cases in our series) and further evaluation such as arteriography is unnecessary. A detailed description on the validity of these signs has been described elsewhere.20 Since no single finding nor combination of findings could be found that reliably diagnosed aortic rupture, an aortogram should be obtained in those cases where clinical suspicion exists and rupture cannot be excluded by the four signs we have enumerated.

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perimental and although no serious side effects have been found, this study will require close follow-up for one to two years. The potential benefit for patients would be close follow-up of their diabetes and retinopathy including retinal photographs and the possible prevention of a serious and disabling complication of diabetes. The trial is in a double-blind format. Otherwise healthy patients with either type I (juvenile) or type II (adult) diabetes are being sought. Patients either without retinopathy or with non-proliferative retinopathy would qualify. Patient referral may be made by calling or having the patients call the Diabetes Center at (317) 630-6374.

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Resources for Treatment of Arthritis in Indiana

MARILYN K. POTTS, M.S.W.¹ KENNETH D. BRANDT, M.D.² Indianapolis

EARLY 36 MILLION PEOPLE in the United States have arthritis or a related musculoskeletal disorder. Despite the high prevalence of rheumatic disease, misperceptions about the cause of arthritis, and its treatment, are widely held by the general public. In addition, the adequacy of rheumatology education for physicians, nurses, and physical and occupational therapists has been questioned. 57

In view of the high prevalence of arthritis, the extent of misinformation about arthritis held by the general public, and the apparent inadequacy of rheumatology content in many medical training programs, it is evident that the need for arthritis related care and education programs is great. Although the Graduate Medical Education National Advisory Committee⁸ has suggested that the United States will experience a surplus of rheumatologists

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Using extant data, we have analyzed the distribution of arthritis care resources in Indiana by tabulating the number of primary care physicians, rheumatic disease specialists, hospital-based physical and occupational therapy departments, and home health agencies, and have performed a synthetic estimate of the prevalence of musculoskeletal disease in each of the state's 92 counties.

The estimated prevalence of musculoskeletal disease by county ranged from 134 to 192 per 1,000 population. Two counties have no primary care physician, while the remainder have from 0.30 to 3.89 primary care physicians per 1,000 adults with arthritis. Twenty-two full-time equivalent rheumatic disease specialists currently practice in Indiana (0.40 per 100,000 population), and the median waiting period for a new patient to obtain a non-emergency appointment with a rheumatic disease specialist is 28 days. While physical and occupational therapists and home health services are readily available throughout the state, we estimate that some 80,000 Indiana residents with arthritis (10% of the total arthritis population of the state) currently reside more than one hour's drive from a rheumatic disease specialist.

by 1990, today, in many regions of the country, a shortage of arthritis specialists exists. In such regions, in which resources for implementation of arthritis-related programs may be limited, efficient use of available rheumatology manpower is especially important.

The development of qualitatively and quantitatively optimal health care resources for people with arthritis throughout Indiana is a major goal of the Indiana University Multipurpose Arthritis Center. In this report we present data from an analysis of the prevalence of musculoskeletal disease, and the quantity and distribution of arthritis care resources, throughout the state. The results have important implications with respect to 1) prioritization of regions of greatest need for arthritis outreach programs, and 2) identification of areas warranting more detailed assessment of health status and manpower needs.

Methods

The most current extant data available were obtained from a variety of sources:

To estimate the prevalence of arthritis in each of Indiana's 92 counties, we multiplied the proportion of county residents in each of four age groups (under 17, 17-44, 45-64 and over 64 years old), according to the 1980 United States census,14 by the corresponding prevalence rate for musculoskeletal disease for each age category (4, 78, 352 and 536 per 1,000 population, respectively), according to the 1980 National Health Interview Survey.1 The resulting products were summed to determine the arthritis prevalence rate per 1,000, thus taking into account the age distribution in each county.*

Information concerning the distribution of Indiana's primary care physicians was obtained from the American Medical Association's 1982 "Directory of Physicians in the United States." ¹⁵ Primary care physicians were defined as general practitioners, specialists in family practice or internal medicine, and pediatricians. Only physicians engaged currently in direct patient care were included. Physicians were excluded from the analysis if they were residents, retired, disabled, otherwise inactive, administrators, medical educators or researchers.

The American Hospital Association's 1983 "Guide to the Health Care Field" provided information concerning the distribution of hospitals with physical and/or occupational therapy departments. Data regarding home health agencies (defined as organizations, e.g., hospital-based home care programs, visiting nurse associations, proprietary home care agencies, which were licensed as of December 1983 by the state of Indiana) were obtained from the Division of Nursing, Indiana State Board of Health.¹⁷

The 1984 physician referral list from the Arthritis Foundation, Indiana Chapter, was used to tabulate the distribution of Indiana's rheumatic disease specialists. These physicians include both board-certified or boardeligible rheumatologists, and primary care physicians whose practices are devoted at least 50% to rheumatology. Eight of Indiana's rheumatologists are staff members of the Indiana University School of Medicine, including one pediatric rheumatologist who treats adults as well as children. Because of their involvement in research, teaching and/or administrative activities, these eight physicians spend an average of 25% of their time (based on a 40-hour work week) in direct patient care. Thus, they were counted, in the aggregate, as two full-time equivalents (FTEs). Arthritis Foundation chapters from adjacent states (Illinois, Kentucky, Ohio and Michigan) provided in-

Figure: Indiana counties with at least one rheumatic disease specialist, and counties lacking a rheumatic disease specialist whose center is more than one hour's drive from the periphery of a county containing such a specialist. The numeral within the circle depicts the number of full-time equivalent rheumatic disease specialists in that county. The arrows extending across the Indiana boundary depict counties situated within one hour's drive of a rheumatologist in the adjacent state of Ohio. Includes board-certified or board-eligible rheumatologists and primary care physicians whose practices are devoted at least 50% to rheumatology.

formation concerning rheumatic disease specialists practicing in areas bordering Indiana. Although some of these physicians are affiliated with medical schools, we did not attempt to estimate the proportion of their time spent in direct patient care.

Each rheumatic disease specialist practicing in Indiana was contacted to ascertain the number of days a new patient would have to wait to obtain a non-emergency appointment. In addi-

Michigan City East Chicago **(2)** • Hammond South Elkhar Gary (2) Bend \III**②** Fort Wayne ന 2 Marion . Lafavet An-Muncie derso . e Richmond • 1 (10) Dayton, Ohio apolis Terre Haute Cincinnati Columbi Ohio 3 looming ton New Albany Rheumatic disease specialist County with no rheumatic disease specialist within a one-hour drive

^{*}In determining the "arthritis prevalence rate," the four most relevant diagnostic categories from the National Health Interview Survey were utilized: arthritis, not otherwise classified; rheumatism; gout; and synovitis, bursitis and tenosynovitis.

tion, we explored the accessibility of rheumatology care in Indiana with respect to the distance patients must travel to obtain the services of a rheumatic disease specialist, physical therapist or occupational therapist. Counties were dichotomized, based on whether the county center was situated more than an estimated one-hour's drive (i.e., outside of a radius of 45 miles) from the periphery of a county containing a rheumatic disease specialist, or hospital-based physical or occupational therapy department, respectively.

Urban and rural counties were analyzed separately with respect to arthritis prevalence rates, and the distribution of primary care physicians and rheumatic disease specialists. Counties located in Standard Metropolitan Statistical Areas (SMSAs), as defined by the Census Bureau, 18 were considered urban; all others were considered rural. One-tailed tests were used for all statistical analyses.

Results

Arthritis prevalence. The mean arthritis prevalence rate for Indiana's 92 counties was 164 per 1,000 population (Standard Deviation [SD] = 13). Rates per 1,000 ranged from 134 to 192, a difference of 43%. Because rural counties contained a higher proportion of people over 64 years old than urban counties (12.38% and 10.65% of the total, respectively, t = 3.98, p < 0.001), rural counties tended to have higher arthritis prevalence rates, based on the methods employed herein. (Notably, the arthritis prevalence rate in people who are over 64 years old is some 50% higher than that in people 45-64 years old, and sometimes higher than the rate for those 17-44 years old). Thus, the mean arthritis prevalence rate for rural counties in Indiana was 167 per 1,000, while that for urban counties was 158 per 1,000 (t = 3.18, p < 0.01).

Arthritis prevalence rates for 17 counties were ≥ 1 SD below the mean for the state as a whole (i.e., 164 per 1,000 population), while rates for 18

TABLE 1
Rheumatic Disease Specialists Per 100,000 Population,
by Standard Metropolitan Statistical Area (SMSA)^a

SMSA	Number of Rheumatologists	Population	Specialists per 100,000 Population
Anderson	0	139,336	0.00
Bloomington	0	98,785	0.00
Chicago-Gary-			
Kenosha ^b	93	7,103,624	1.20
Cincinnati, OH,			
KY-IN ^e	11	34,291	0.65
Elkhart	0	137,330	0.00
Evansville, IN-KY ^d	1	268,559	0.37
Fort Wayne	2	382,961	0.52
Indianapolis	17^{e}	1,166,575	1.45^{e}
Kokomo	0	103,715	0.00
Lafayette-			
West Lafayette	2	121,702	1.64
Louisville, KY-IN ^f	10	906,152	1.10
Muncie	0	128,587	0.00
South Bend	2	280,772	0.71
Terre Haute	0	176,583	0.00

 $^{^{4}\}mathrm{SMSAs}$ located entirely or partly within Indiana, according to U.S. Census Bureau. 18

counties were ≥ 1 SD above the mean. Two counties had arthritis prevalence rates ≥ 2 SD below the state mean (135 and 134 per 1,000, respectively), while the rate for one county was ≥ 2 SD higher (192 per 1,000) than the mean rate for Indiana as a whole.

The total number of people with arthritis in Indiana was estimated to be 844,907 (15% of the state's 1980 population of 5,490,224). This included 6,053 children under 17 years old. By county, the estimated number of individuals with arthritis ranged from 886 to 132,548.

Primary care physicians. Indiana contains 2,167 practicing primary care

physicians (0.39 per 1,000 population), including 247 pediatricians. For Indiana as a whole, the rate of primary care physicians per 1,000 people with arthritis is 2.45. However, because the arthritis prevalence rate among individuals under 17 years old is low (0.04 per 1,000, according to the National Health Interview Survey), the ratio of pediatricians to arthritic children is much higher than the ratio of other primary care physicians to arthritic adults. Since inclusion of pediatricians and children with arthritis inflates the estimate of primary care physicians per arthritis population, results reported hereafter are based exclusive-

^bIncludes Lake and Porter Counties in Indiana.

^{&#}x27;Includes Dearborn County in Indiana.

^dIncludes Gibson, Posey, Vanderburgh and Warrick Counties in Indiana.

cine spend an average of 25% of their time in direct patient care, they were counted, in the aggregate, as two full-time equivalents (FTEs). Accordingly, the number of FTE rheumatic disease specialists in the Indianapolis SMSA is 11, or 0.94 per 100,000 population.

Includes Clark and Floyd Counties in Indiana.

TABLE 2
Arthritis Prevalence and Availability of Primary Care Physicians in Counties with No Rheumatic Disease Specialist^a

County			Primary Care Physicians per 1,000 Adults with Arthritis ^b
Vigo	19,526	174	2.21
Knox	7,759	186°	1.81
Lawrence	7,343	173	2.47
Miami	5,961	150	1.69
Jackson	6,075	166	2.65
Greene	5,581	184°	2.70
Daviess	4,950	178°	1.42
Clay	4,627	186°	1.09^{e}
Sullivan	4,060	$192^{\rm d}$	$0.74^{\rm e}$
Decatur	3,860	162	1.83
Jennings	3,392	148	1.78
Perry	3,364	174	2.09
Owen	2,769	175	$1.09^{\rm e}$
Martin	1,763	160	$1.14^{\rm e}$
TOTAL	81,030		

^aNo rheumatic disease specialist located within one hour's drive from center of county. See text for details.

ly on analyses which exclude pediatricians and children with arthritis.

The state-wide rate of primary care physicians per 1,000 adults with arthritis is 2.30. By county, this rate varies widely (range = 0.00-3.89, x = 1.97, SD = 0.72). The mean rate for urban counties is 2.16 (SD = 0.75), while that for rural counties is 1.85 (SD = 0.69)(t = 1.93, p < 0.05). Sixteen counties have rates \geq 1 SD below the state's mean of 2.30 per 1,000 adults with arthritis, while those for 10 counties are ≥ 1 SD above the mean for Indiana. Three counties have rates $\geq 2 \text{ SD}$ below Indiana's average, while two have rates ≥ 2 SD above the state mean.

Home health care and physical and occupational therapy services. Indiana has 93 licensed home health agencies, whose combined service areas encompass the entire state. Each agency provides skilled nursing services; 84 (90%) also employ home health aides.

The state contains 105 hospitals with physical therapy departments and 45 with occupational therapy departments. Each county has at least one physical therapy department, or is adjacent to a county in which hospital-based physical therapy services are available. In addition, each county has at least one occupational therapy department, or the county's center is situated within an hour's drive of the

periphery of a county containing a hospital-based occupational therapy department.

Rheumatic disease specialists. Twenty-eight rheumatic disease specialists currently practice in Indiana. The number of FTE rheumatic disease specialists in Indiana is 22, or 0.40 per 100,000 population.

Since half of Indiana's FTE rheumatic disease specialists (0.94 per 100,000 population) practice in the Indianapolis SMSA, only 11 (0.31 per 100,000 population) are situated in the remainder of the state (*Table 1*). Notably, six SMSAs (Anderson, Bloomington, Elkhart, Kokomo, Muncie and Terre Haute), with a combined population of 784,336, have *no* rheumatic disease specialist.

Figure 1 depicts the counties in which Indiana's rheumatic disease specialists practice, and those in which such specialists are not readily accessible. Although Wayne, Fayette and Union Counties do not possess a rheumatic disease specialist, residents of these counties are within an hour's drive of rheumatologists in Dayton, Ohio. Similarly, people in Ripley, Dearborn, Ohio and Switzerland Counties reside within an hour's drive of rheumatologists in Cincinnati, Ohio. The data thus indicate that an estimated 81,030 people with arthritis, residing in 14 Indiana counties, must drive more than one hour to obtain the services of a rheumatic disease specialist (Table 2). This represents 10% of Indiana's estimated arthritis population.

Among Indiana's 28 rheumatic disease specialists, the waiting period for a routine appointment for a new patient currently ranges from 2-95 days (median = 28, mean = 30, SD = 22 days). Thirteen of these physicians (nine in the Indianapolis SMSA and four in the remainder of the state) have waiting periods \geq 30 days. For the 17 specialists in the Indianapolis SMSA, the median waiting period is 30 days (mean = 30, SD = 20 days), while for the 11 practicing in the remainder of the

^bIncludes general practitioners and specialists in family practice or internal medicine. Excludes pediatricians and children with arthritis < 17 years old.

^c≥ 1 SD above state mean of 164 per 1,000 population.

 $^{^{\}rm d} \ge 2$ SD above state mean of 164 per 1,000 population.

^e ≥ 1 SD below state mean of 1.97 per 1,000 adults with arthritis.

state it is less than half as long (median = 14, mean = 29, SD = 26 days). Although each staff rheumatologist at the Indiana University School of Medicine sees new outpatients only one day per week, the median waiting period for these physicians is similar to that for the other rheumatic disease specialists in the Indianapolis SMSA (median = 30, mean = 27, SD = 9 days).

Discussion

Based on our analyses of these extant data, the 14 Indiana counties in which a rheumatic disease specialist currently is not readily accessible might appropriately be designated as priority areas for various types of arthritis-related outreach programs. The results, therefore, are relevant to the mission of the Indiana University Arthritis Center. Those counties among the 14 which have arthritis prevalence rates higher than Indiana's average (i.e., Knox, Greene, Daviess, Clay and Sullivan Counties) may be especially important targets for lay and/or physician education programs (Table 2). For continuing education ef forts directed at nurses and allied health professionals, it may be appropriate to prioritize further those counties among the 14 (i.e., Clay and Sullivan Counties) which have relatively few primary care physicians per capita and high arthritis prevalence rates (Table 2).

Our results indicate that physical therapists, occupational therapists and home health services are readily available throughout Indiana. However, rheumatic disease specialists tend to be centralized within the Indianapolis SMSA. The number of primary care physicians per capita, and per population with arthritis, varies widely from county to county. Notably, more primary care physicians per capita practice in urban than in rural areas, while arthritis prevalence rates tend to be higher in rural rather than urban areas.

Four points should be made regarding the methods employed herein.

First, interviewers for the National Health Interview Survey, from which our arthritis prevalence data were derived, record a specific diagnosis only if the respondent has been given one by a physician, or if the symptoms reported by the respondent lead to an unambiguous diagnosis. Otherwise, rheumatic conditions are categorized as "arthritis, not otherwise classified." Our inclusion of the latter category. which is based entirely on selfdiagnosis, may have produced an inflated estimate of the prevalence of arthritis. Accordingly, our results concerning the relative prevalence of arthritis throughout Indiana's counties may be more valid than the absolute figures for each county.

Second, although quantitative data are presented concerning the regional distribution of Indiana's primary care physicians, physical and occupational therapists, and home health services, no attempt was made to ascertain knowledge/skill levels with respect to management of patients with arthritis.

Third, our criterion for analyzing geographic accessibility to health care providers is arbitrary (i.e., an hour's driving time, based on the distance between each county's center and the periphery of a county containing a therapist or rheumatic disease specialist). Other measures, e.g., linear distance, travel distance and travel cost, 19 might have yielded different results.

Finally, it should be emphasized that our results cannot be utilized to formulate a statement concerning Indiana's rheumatologic manpower needs. Although the state, as a whole, clearly does not meet the Graduate Medical Education National Advisory Committee's standard of 0.7 rheumatologists per 100,000 population, the International League Against Rheumatism's recommendation of 1 practicing rheumatologist per 100,000,9 or the 4.2 per 100,000 rate suggested by the Arthritis Foundation,20 we have made no attempt to ascertain for Indiana the appropriate number of

rheumatic disease specialists per population. Neither have we attempted to determine the proportion of people with arthritis who require treatment by a physician,²¹ or who might benefit more from a specialist's services than from those of a non-specialist.

An adequate assessment of the above issues would require examination of several factors, including local referral patterns;12,22,24 the number of visits made per year by arthritis patients to specialists and non-specialists, and the average duration of each visit;12,22,25 the current use of physician extenders;22 the degree to which primary care physicians and community-based allied health professionals are skilled in arthritis treatment; and health status outcomes in patients treated by rheumatologists compared to those treated by primary care physicians. Nonetheless, the rapid, generalizable technique which we have utilized herein will enable us to prioritize relative areas of need for arthritis-related outreach programs in Indiana, and to identify particularly relevant regions in which to conduct in-depth analyses of the above factors.

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erse Reactions: Muscle cramps, weakness, dizziness, headache, dry
th; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatcical conditions; nausea and vomiting, diarrhea, constipation, other
trointestinal disturbances; postural hypotension (may be aggravated by
hol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias,
rus, pancreatitis, xanthopsia and respiratory distress including pneuritis and pulmonary edema, transient blurred vision, sialadenitis, and
go have occurred with thiazides alone. Triamterene has been found in
1 stones in association with other usual calculus components. Rare
Jents of acute intersitifial nephritis have been reported. Impotence has
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Autoimmune Hearing Loss

JAMES E. GAMBLE, M.D.
JOHN A. BIZAL, M.D.
STEPHEN C. FERGUSON, M.D.
EDWARD DAETWYLER, M.D.
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UTOIMMUNE DISEASES may affect virtually every system in the body. Very little has been written concerning their effect on the ear. Although rarely considered, when studying an autoimmune process, sensorineural hearing loss may present as an initial finding in an autoimmune disease. If a sensorineural hearing loss develops gradually over a period of one week to several months, and there is no obvious etiology, consideration should be given that this is possibly the first sign of onset of an autoimmune process. If after a complete audiological evaluation the cause for the hearing loss is not apparent, evaluation for an autoimmune disease should be done.

The hearing loss associated with an autoimmune process is a treatable entity and the results are better when begun early.

The ear may be catastrophically affected by the immune system. An example of this is the following case:

A 58-year-old woman had been completely well until one week prior to her initial office visit. At that time she developed a gradual onset of pulsating tinnitus in the left ear and hearing loss. Examination revealed normal tympanic membranes, no fluid and, on audiometric examination, relatively good hearing with a 5 to 10 decibel conductive loss in both ears. No specific treatment was given.

One month later she presented with

From Tri State Otolaryngology, Head and

much worse hearing which she said had been gradually progressive during the past month. She had had no ear pain or vertigo. On examination the tympanic membranes were thickened and an audiometric evaluation found a mixed hearing loss of approximately 75 decibel in the right ear and 40 decibel in the left ear.

Two weeks following this visit tympanostomies with tubes were performed. The tympanic membranes were extremely thickened, leaving little middle ear space. The tympanic membranes were primarily thickened in the middle (fibrous layer) and appeared like a watery, edematous swelling. There was a small amount of straw colored fluid in the remaining middle ear space. This procedure provided no improvement. Within two months of the onset of symptoms the patient had severe hearing loss bilaterally with conductive and sensorineural components.

Two weeks later there was even further progression of hearing loss and by this time a drainage had developed in both ears, which failed to produce any growth on culture.

At this time, even though the patient was otherwise symptom free, it was decided to evaluate her for autoimmune disease, since all other known causes had been ruled out. Biopsies of the kidney and skin produced a positive diagnosis of periarteritis nodosa. She was placed on steroids with almost immediate improvement.

The drainage ceased, the perforations healed and the tympanic membranes reverted to normal appearance. The hearing improved by 10-20 decibels. However, on examination one year later, the patient continued to have a moderate mixed type hearing loss, although with no evidence of progression.

Autoimmune sensorineural hearing loss was described by Brian McCabe

in 1979. His conclusions were that this type of deafness usually develops over a period of weeks to months, not suddenly or over a period of years. All of his 18 patients responded to dexamethasone and cyclophosphamide therapy given over a period of eight months to two years.

The cause and site of lesions are not exactly clear. Leone, et al.² found evidence of involvement of the fenestrated filtering capillaries of the endolymphatic sac, but found no involvement of the unfenestrated capillaries of the remainder of the cochlea. They believed this may be related to the deposition of antigen antibody complexes in these filtering capillaries, the same as may occur in the filtering capillaries of the kidney.

T.J. Yoo and associates³ were able to produce hearing changes in the brain stem evoked response in rats and mice immunized with native type II collagen. Pathological findings in these animals was vacuolate degeneration of the cochlear neurons throughout most of the cochlea.

In our case these were changes in the tympanic membrane, middle ear and inner ear consistent with a vasculitis. This would be in agreement with McCabe's findings.

In summary, a patient was described with an autoimmune sensorineural hearing loss. The etiology of the hearing loss in most cases appears to be related to a vasculitis but this may not be the situation in all instances.

A patient who presents with a mixed or pure sensorineural hearing loss, which has been present for a duration of a week to a few months, in which no other etiology is apparent, should be suspected of having an autoimmune disease, even though no other signs or symptoms are present. This is a treatable problem and will respond to appropriate therapy.

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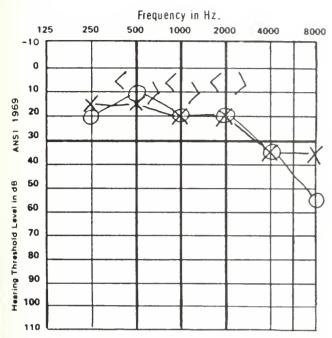


FIGURE 1: Audiogram taken at the time of the initial visit. (X = left ear; 0 = right ear)

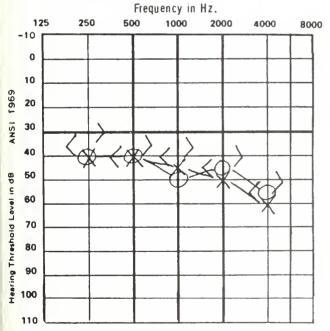


FIGURE 3: Audiogram shows a severe mixed hearing loss six weeks after onset, prior to steroid therapy.

1. McCabe BF: Autoimmune sensor-

ineural hearing loss. Ann Otol,

Leone CA, et al: Endolymphatic sac:

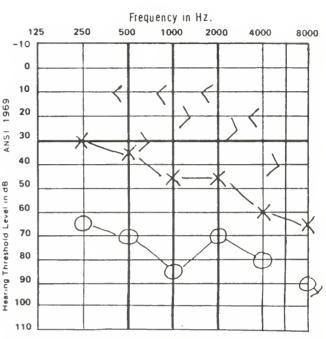


FIGURE 2: A further loss is noted in this audiogram, taken on the second visit one week later.

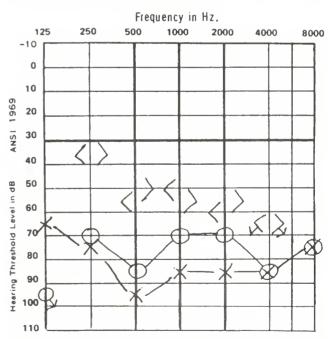


FIGURE 4: Audiogram demonstrates the improved hearing, although not normal, after six weeks of prednisone therapy.

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Skin Diseases: Current Concepts, Therapy

3. INFLAMMATION

BRIAN POTTER, M.D. Michigan City

NFLAMMATORY DISEASE of the skin is more common in men than women because of the differential incidence of occupational contact dermatitis, which represents at least 45% of all industrial disorders. This type of dermatitis is caused by exogenous influences, including defatting solvents, detergents, soaps, primary irritants, and contacted allergens.

During the last 20 years the existence of a third system in the epidermis, besides the keratinocytes and melanocytes, has been demonstrated. These are the Langerhans cells, which are a vital factor in mediation of allergic contact sensitivity. They take up metallic salts (and thus can be demonstrated histochemically), and present these potential antigens to circulating sensitized T-lymphocytes.²

Allergic contact dermatitis represents an acquired, indirect reaction to a sensitizing agent. It is an immunological response of the type due to the development of cellular immunity. The most common allergens include poison ivy, nickel, chromate ions, other chemicals, dyes, fragrances and other ingredients of cosmetics. Sen

sitizers are even found in over-thecounter products, toothpastes, lubricating ointments, hair preparations and rubber gloves.³

Several types of eczematous dermatitis represent inflammation of endogenous origin. Atopic dermatitis is an inflammatory disease affecting 2-3% of all children between the ages of 1 and 5, and 0.7% of persons of all ages, often persisting (in 60%) for 15-20 years. The cause is unknown but it is familial. It is associated with the familial traits of seasonal rhinitis, asthma, raised levels of IgE in the serum, decreased cell-mediated immunity and decreased chemotaxis. Besides the immunologic differences, however, the sympathetic branch of the autonomic nervous system is abnormal in children with chronic atopic dermatitis. Specifically, their betasympathetic-mediated responses are impaired.4 This type of dermatitis is also associated with the precocious development of ocular cataracts, and with the common complication of secondary infection.

The entire mammalian class seems to be subject to seborrheic dermatitis, which occurs in humans on the scalp, face, ears and chest, the parts of the body where hair follicles and sebaceous glands are larger than elsewhere. These sites are heavily populated by a nonpathogenic organism, Malazzezia ovalis, which may be incriminated in the pathogenesis of seborrheic dermatitis. Some cases of this dermatitis have been cleared by a topical fungicide, specifically 2% ketoconazole cream.5 Other chemicals effective in seborrheic dermatitis, including sulfur, selenium sulfide, and zinc pyrithione, are also active against M. ovalis.

The characteristic symptom of most types of dermatitis is pruritis, which provokes the scratch reflex. In susceptible persons, repeated rubbing and scratching leads to reactive hyperplasia of the epidermis, known as neurodermatitis. In chronic cases, the original cause is often no longer discernible. These states of hypersensitivity of the skin are referred to as eczema. This term bears the same relationship to dermatitis as pneumonia does to pneumonitis. Eczema is characterized morphologically by erythema, the eruption of papules, vesiculation and exudation in acute cases, and the formation of papular plaques, thickening of the skin and scaling in chronic cases. The histologic changes common to all forms of eczematous dermatitis are edema of the epidermis and perivascular infiltration of inflammatory cells in the dermis.

Topical emollients are moderately effective in therapy for eczematous dermatitis. No nonsteroidal anti-inflammatory agents can be recommended, except coal tar for chronic conditions. All forms of dermatitis are treated by the topical application of corticosteroids in creams and ointments.

The glucocorticoid receptor in human skin is a specific, steroid-binding, protein molecule. Corticosteroids applied topically alter the number of immune-active Langerhans cells and their function. Macrophages are profoundly affected, thus depressing local mechanisms active in defense and immune reactions.

The antiproliferative effects of cor-

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ticosteroids include both inhibition of epidermal mitosis and decrease in the rate of synthesis of DNA. Keratinization is thus returned toward the normal, and the fibrous stage of inflammation is lessened by decreased regeneration of fibroblasts.

Suspensions of corticosteroids are used for intralesional injection. Several inflammatory skin diseases can be treated in this way, including localized eczema, discoid lupus erythematosus, sarcoidosis, lichen planus, and benign lymphocytic infiltrates. Triamcinolone is supplied for this purpose as acetonide 10 mg/ml (Kenalog), hexacetonide 5 and 20 mg/ml (Aristospan) and diacetate 25mg/ml (Aristocort), methylprednisolone acetate 20 mg/ml (Depomedrol), dexamethasone acetate 8 mg/ml (Decadron-LA) and betamethasone sodium phosphate and acetate 6 mg/ml (Celestone soluspan).

Topical steroids have little effect in allergic eczematous contact dermatitis. This is one of the few examples of dermatitis for which systemic administration is appropriate therapy. However, systemic steroids are indicated for some other conditions including severe drug eruptions and blistering diseases.

Pemphigus vulgaris and bullous pemphigoid are examples of a second type of immunological disorder in which cytotoxic antimembrane antibodies circulate in the blood. In both diseases, the antigens are synthesized by the epidermal cells. In pemphigus, the autoantibodies are contained within IgG fractions in patients' serum.8 The antibody-antigen reaction releases an enzyme or enzymes that act on the intercellular substance of the epidermis and mucous membrane, causing dissolution of intercellular attachments, resulting in large blisters and erosions. This grave disease is associated with the inheritance of HLA antigen DRW4, which occurs in 55% of pemphigus patients.9 Patients with this disease may require 80 mg daily of prednisone, or more. However, patients may have prolonged remission after successful therapy, and maintenance doses of steroids may not be required.¹⁰

In pemphigoid, circulating antibodies are directed against the epidermal basal cell membrane or cytoplasm or both, with deposition of IgG and complement in the basement membrane zone. Again there is an indirect release of enzymes, leading to loss of adherence of the epidermis, and deep blistering. Pemphigoid is associated with another, probably autoimmune, disease, rheumatoid arthritis. Pemphigoid is associated with another in the circular probably autoimmune, disease, rheumatoid arthritis.

Erythema multiforme, necrotizing vasculitis and systemic lupus erythematosus are examples of a third type of immunologic reaction, in which immune complexes are circulating and become deposited in the cutaneous microvasculature. Erythema multiforme often represents a drug eruption, but is also associated with infections, malignancy, physical factors, contact reactions, endocrine disorders and collagen disease. IgM and complement are found in blood vessel walls in the papillary dermis.

Allergic vasculitis is caused by drugs and infections, particularly penicillin and sulfonamides, streptococcal infection and hepatitis. The prototype of this disorder is hepatitis B virus urticaria, which occurs at the same time as serum hypocomplementemia and the circulation and deposition of immune complexes. The complexes are composed of viral antigen, antibody and complement.²

In lupus erythematosus, immunoglobulins and complement are deposited at the dermal-epidermal junction in areas of skin that are exposed to light. This is a heterogeneous disease or group of diseases, ranging from the chronic, scarring, localized type to the classic, disseminated type. However, systemic lupus is definitely related to a peculiar, atypical, photosensitive, ANA-negative, subacute, dermatological lupus with large, annular and polycyclic lesions.2 These various subsets of lupus vary in prevalence of renal disease, and therefore in prognosis.14 Laboratory

tests that may help in the categorization of cases of lupus include CBC, urinalysis, sedimentation rate, blood urea, creatine clearance, reagin test, serum protein electrophoresis, ANA, anti-DNA, anti-ENA (extractible nuclear antigens, ribonucleoprotein and thymus extract), complement, rheumatoid factor, Coombs test, cryoglobulins and urinary light chains.¹⁵

Many of the changes observed in inflammation are accounted for by the release of histamine, which appears in the interstitial fluid during the development of edema. The histamine is released from mast cells and basophils. When this is mediated by IgE, each molecule of antigen bridges two adjacent IgE molecules, each of which is attached to a mast cell or basophil. This activation results in extrusion and dissolution of the cellular granules, to liberate histamine and heparin. This is manifested by the rapid development of acute urticaria, a sudden eruption of circumscribed, raised, erythematous, superficial, edematous weals.

This fourth type of immunological reaction occurs in atopic subjects, in anaphylaxis and serum sickness, contacts with chemicals, animals and plants, and some drug eruptions. Another drug-induced urticaria is caused by the direct liberation of histamine from the cell by the drug. Nonimmunologic urticaria due to the direct release of mast cell granules can be caused by physical pressure, especially in urticaria pigmentosa and other forms of mastocytosis. Acute urticaria is associated with other physical causes, including cold, heat, light, water contact and vibration.16 The commonest type of urticaria due to heat is cholinergic, apparently an hypersensitivity to acetylcholine, and therefore occurs on exercise and excitement as well as from heat.

Chronic urticaria occurs from the causes already enumerated and sometimes from underlying systemic disease. In as many as 70% of cases,

however, the cause remains undiscovered.

Patients with urticaria should have a number of tests to exclude possible causes. Blood count, sedimentation rate, urinalysis and chest x-ray should be done as a minimum, and perhaps serum bilirubin, complement, rheumatoid factor, ANA, and cryoglobulins, and sinus and dental xrays. However, unless there are suggestive findings in the history or physical examination, laboratory studies are not likely to be helpful in the evaluation of chronic urticaria. 17

In treatment of urticaria, the dose of antihistamines should be increased to tolerance, or until the itching is suppressed, whichever is lower. Conventional antihistamines block H1 receptors; the addition of the selective H2 receptor antagonist cimetidine has no advantage in the treatment of urticaria.18 As in any benign chronic condition, systemic corticosteroids are to be avoided.

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Look-Alike and Sound-Alike **Drug Names**

BENJAMIN TEPLITSKY, R. PH. Brooklyn, N.Y.

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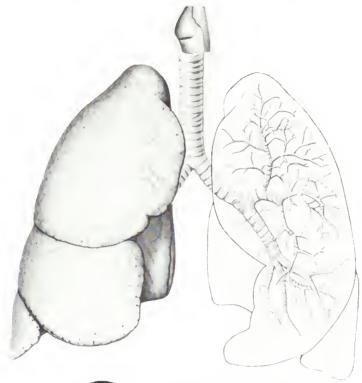
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Streptococcus pneumoniae (Diptococcus pneumoniae) Haemoph
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ment should include sigmoidoscopy appropriate bacteriologic studies and Illuid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been updated in the studies of the studie

produced by C difficile. Other causes of colitis should be ruled out.

Precaulions. General Precautions — It an allergic reaction to Cector's releafoin Lityly occus the drug should be discontinued, and, if necessary the patient should be treated with appropriate agents e.g. pressor aimnes aninistamines or corticosteroids. Prolonged use of Declor may result in the overgrowth of monsusceptible organisms. Carelui observation of the patient is essential. If superinterion occurs during therapy, appropriate measures should confident in the overgrowth of the patient is essential. If superinterion occurs during therapy, appropriate measures should confident in the overgrowth of the patient in the overgrowth of the overgrowth ove

colitis

Usage in Pregnancy — Pregnancy Category B — Reproduction
studies have been performed in mice and rafs at doses up to 12
times the human dose and in feirets given three times the maximum

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Usage in Children — Safely and effectiveness or trins product in use in initiatis less than one month of agin have not been established Adverse Reactions. Adverse effects considered related to therapy with Cector are uncommon and are listed below. Gastromtestinal symptoms occur in about 2.5 percent of patients and include diatrihed. In 1 in 70). Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and somitting have been reported rarely. Hyper constitutions are stated to the state of th

the syndrome Cases of anaphylaxis have been reported, half of which have

occured in patients with a history of penicilin allergy Other effects considered related to therapy included econophilia! in 50 patients, and gental pruntus or vaginitis (less than 1 in 100 patients). Causal Relationship Uncertain — Transitivy abnormalities in climical laboratory test results have been reported. Although they were of uncertain ectology they are listed believe to serve as alerting information for the physician. Hepatics (Sight elevations in SGOT SCPT or alkaline Hepatics (Sight elevations in SGOT SCPT or alkaline Hematoporetic. — Transient fluctuations in leukocyte count predominantly imphocytosis occurring in inatists and young children (1 in 40). Renail — Slight elevations in BUN or serium creationine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

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Contraindications: Severe left ventricular dysfunction (see Warnings), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (except in patients with a functioning artificial ventricular pacemaker), 2nd- or 3rd-degree AV block. **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction < 30% or moderate to severe symptoms of cardiac failure) and in patients with any degree of ventricular dysfunction if they are receiving a beta blocker. (See *Precautions*) Patients with milder ventricular dysfunction should, if possible, be controlled with optimum doses of digitalis and/or diuretics before ISOPTIN is used (Note interactions with digoxin under *Precautions*.) ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild and controlled by decrease in ISOPTIN dose). Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported Such elevations may disappear even with continued treatment, how-ever, four cases of hepatocellular injury by verapamil have been proven by re-challenge. Periodic monitoring of liver function is prudent during verapamil therapy Patients with atrial flutter or fibrillation and an accessory AV pathway (e.g. W-P-W or L-G-L syndromes) may develop increased antegrade conduction across the aberrant pathway bypassing the AV node, producing a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion, which has been used safely and effectively after ISOPTIN Because of verapamil's effect on AV conduction and the SA node, 1° AV block and transient bradycardia may occur. High grade block, however, has been infrequently observed. Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high morof serious adverse effects were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. Most adverse effects responded well to dose reduction and only rarely was verapamil discontinued Precautions: ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, close surveillance of vital signs and clinical status should be carried out. Combined therapy with ISOPTIN signs and clinical status should be carried out. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patients should be carefully monitored to avoid over- or under-digitalization. ISOPTIN may have an additive effect on avoid over- or under-digitalization. ISOPTIN may have an additive effect on lowering blood pressure in patients receiving oral antihypertensive agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Clinical experience with the concomitant use of ISOPTIN and short- and long-acting nitrates suggest beneficial interaction without undesirable drug interactions. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumor generous potential, and verapamin was not mutagenic in the Ames test. Pregnancy Category C: There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk, therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block.

3rd degree (0.8%), bradycardia HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported (See *Warnings*.) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred vision, syncope, muscle cramp, shakiness, claudication, hair loss, macules, spotty menstruation **How Supplied**: ISOPTIN (verapamil HCl) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side Revised August, 1984



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Alternative Delivery Systems: An Update on HMOs

A Message from the Executive Director

Significant changes have been taking place recently within the health care delivery system. Increased numbers of providers, changing public policy, new influences from industry and a growing number of health care delivery options are coming together to create changes in health care financing.

What is really new is that industry is playing a key role in shaping health care policy and in making health care decisions. Employers and benefit plan administrators are seeking cost effective alternatives to the traditional delivery of health care.

HMOs Get Boost from Federal Government

The federal government appears to be banking on cost-effective health maintenance organizations (HMOs) to preserve the future solvency of the Medicare program. HMOs began signing up Medicare beneficiaries Feb. 1, 1985. Even prior to the implementation of this new policy, HMOs had been experiencing dramatic enrollment growth. So the opportunity to expand into the Medicare market probably will be an added boon for the HMO industry.

Contrary to government advocacy, however, some private sector purchasers no longer view HMOs as solutions for arresting health care cost escalation. They point to the generous profits some HMOs are accumulating by enrolling healthy workers, and question why premium price competition cannot be stimulated.



DONALD F. FOY Executive Director Indiana State Medical Assn.

Nevertheless, current HMO revenues of \$9.6 billion have been projected to grow to \$70 billion by the end of this decade, according to the market research firm of Frost & Sullivan. Another emerging trend is the growth of non-federally qualified, for-profit HMOs. Currently, about one-fourth of the HMOs in the U.S. are for-profit.

Government support of HMOs began with the passage of the HMO Act in 1973, and really blossomed with the passage of the Tax Equity and Fiscal Responsibility Act (TEFRA) in 1982, in which provisions for HMO enrollment

of Medicare beneficiaries were incorporated. Although the Reagan administration welcomed the addition of prepaid health plans for its costly Medicare population, it spent more than two years working on regulations to ensure that adverse selection and windfall profits for HMOs did not result from the new enrollment opportunities.

HMO advocates cheered the release of a major analysis of HMO costeffectiveness published in the June 7, 1984 issue of the New England Journal of Medicine. That study revealed that expenditures of one HMO in the Northwest are 25% less than for comparable indemnity plans, primarily due to a 40% difference in hospital admission rates. Since the release of the TEFRA regulations with tables of rates that identify, by county, how much an HMO will be paid for each Medicare beneficiary, questions have been raised about the generous payment structure. If HMOs are 25% less expensive than the fee-for-service health care delivery, why should they be collecting 95% of the average Medicare per capita costs that were based on fee-for-service medical costs?

Community Rating and HMO Profits

Employer disenchantment, publicly voiced by a number of multistate corporations, stems from a concern about HMO profiteering. These employers criticize a requirement in the HMO Act for community rating of enrolled groups. HMOs historically have set their premiums based on the actuarial

assessment of the community, although this is starting to change with the growth of non-federally qualified HMOs that are contracting with employers to provide coverage based on the experience of individual company employees.

Some community rated HMOs have been accused of "shadow pricing," that is, setting their premiums just below those of Blue Cross and commercial carriers. The 95% level at which Medicare has elected to pay HMOs represents a "shadow price" of sorts, in that it is linked inextricably to non-HMO utilization experience. Reacting to this concern, government regulations require that any savings resulting from differences between HMO costs and the 95% payment rate be passed along to Medicare beneficiaries, not the HMOs, in lower rates or additional benefits.

By being required under the HMO Act to pay no less than what they would pay for their other insured employees, benefit managers contend that they are generating windfall profits to HMOs that have enrolled the more actuarily attractive workers. At Honeywell, Inc., for example, where 70% of the employees are enrolled in HMOs in Minnesota, the company estimates it is losing \$4 million to \$5 million a year through subsidization of HMOs that have attracted its healthiest workers.

Instead, more employers would like to be able to negotiate their HMO rates, as some are now doing with nonfederally qualified HMOs.

Additional profit margins are bestowed on community rated HMOs that enroll employees availing themselves of worksite wellness programs. The most effective of these programs not only improve morale, reduce absenteeism and increase productivity, they also effect changes in the health care utilization rates from which a work force's experience is actuarily computed. If experience is ignored by HMO rating practices, then wellness program costs incurred by an employer

result in fewer preventive services being provided by the HMO, and, hence, greater profits to the HMO resulting from that segment of its population.

According to critics of community rating requirements, employees who elect to stay in a company's indemnity plan are purported to be older and less fit, leading to greater health care utilization and subsequent increases in the experience rated premium. This phenomenon of adverse selection has not been adequately proven. In fact, there are some examples where HMOs end up drawing in enrollees who are less actuarily attractive, leading to losses rather than profits for these organizations.

Meanwhile, a certain divisiveness within the HMO industry has emerged due to the community rating issue. In Minnesota, HMO industry leaders are pushing to have the requirement for community rating erased from the state's HMO Act.

As investors have been moving into the market, strategic decisions by HMOs not to seek federal qualification have been made, in part because of the community rating constraint and also for profit reasons. For example, an Arizona HMO with the largest proportion of Medicaid enrollees is not federally qualified.

The entrance of HMOs that do not have to comply with community rating standards into some markets, added to the successful moderation of health care cost escalation in some areas, has injected a greater element of competition for HMOs. If employers are able to convince policy makers of the unintended consequences of community rating, it could be raised as part of the reauthorization of the HMO Act.

Equally important to the decision concerning federal qualification is the ability to evade the federally mandated benefits that qualified HMOs must offer. The recent requirement to provide organ transplant coverage may make newer HMOs reconsider the value of federal qualifications. Some for-profit HMOs feel confident that they can com-

pete on price with some of the established HMO giants and capture the attractive private employer group market through benefit and premium negotiations.

Problems with Florida HMO

Problems continue to plague the controversial Florida health maintenance organization, International Medical Centers. Recently, two clinics in St. Petersburg filed for protection under Chapter 11 of the federal bankruptcy law. The clinics provide care for about 3,200 members of IMC's "Gold Plus" plan. The state has been deluged with so many complaints about quality of care, lapses in coverage, and unpaid doctor bills that the state insurance commissioner has proposed a legislative package of tougher industry controls.

Report Card on HMOs

The nation's physicians are becoming increasingly positive about health maintenance organizations, according to a national survey released recently in Washington. Half of the country's physicians say they are favorable about HMOs today compared with 36% who were favorable in 1981, according to Louis Harris and Associates. Harris surveyed the physicians as part of a major examination of attitudes about HMOs. The study, conducted last fall, was commissioned by the Henry J. Kaiser Family Foundation.

"Many of the country's physicians still have deep reservations about health maintenance organizations," said Humphrey Taylor, president of Louis Harris and Associates. "But the trend is clear: Physicians are becoming more and more positive about prepaid plans."

HMOs, which provide subscribers with nearly all of their medical care for a prepaid premium, currently have more than 15 million members across the nation. At the current growth rate, HMO membership will double every five years.

"There is dramatic evidence," Taylor

said, "that the rapidly growing HMO movement has had a significant impact on physicians in recent years."

For example, only 27% of the physicians surveyed in 1981 said they were considering affiliating with an HMO. In 1984, the number considering an HMO affiliation had nearly doubled to 46%.

Moreover, six of 10 physicians who are not part of an HMO or other prepaid plan say they believe prepaid plans will affect their practices over the next 10 years, and 26% believe they will be affected a great deal.

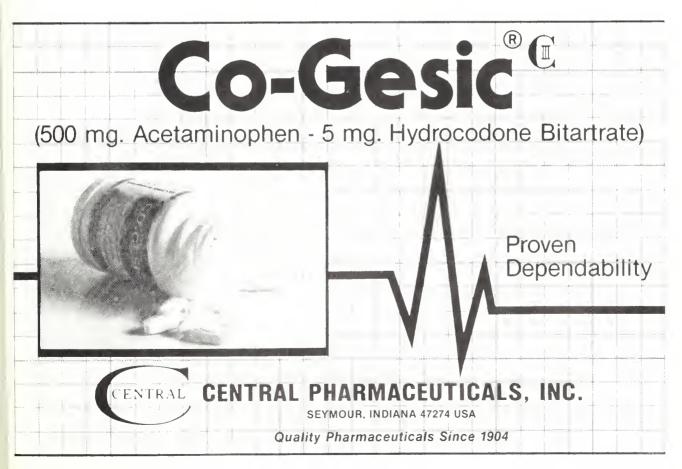
Eighteen per cent of the fee-forservice physicians surveyed say the presence of an HMO in their area has reduced their income, and 14% say they have reduced either the number or duration of hospital stays among their patients because of the prepaid groups operating in their area. "By a great majority – 78% – physicians believe HMOs are effective in containing health care costs," Taylor said. "But about two-thirds also believe that the cost-containment incentive causes HMOs to lower the quality of care to an unacceptable level."

Taylor explained that the share of physicians who believe HMOs offer lower quality care than traditional systems has changed little since 1981. He added that many physicians believe that HMOs perform fewer lab and diagnostic tests than may be necessary, employ less qualified doctors and do not allow for adequate doctor-patient relationships.

"There is no evidence in the survey that HMOs employ physicians of lower caliber than the physician community as a whole as measured by board certification and length of practice," Taylor said. "And, while the research didn't address the other areas of concern specifically, the survey did reveal that HMO subscribers are well pleased with the medical care they receive."

When it comes to their own satisfaction, physicians in prepaid plans and physicians in traditional fee-for-service practices show comparable levels. However, physicians in HMOs and other prepaid groups are more satisfied with three areas of practice: "professional peer support," "affiliations with a major medical center," and "time available to devote to nonprofessional interests, family and friends."

"The high levels of satisfaction with prepaid practice are underscored by the fact that 86% of the prepaid group physicians plan to continue in prepaid practice," Taylor concluded.



Rising Health Costs and Challenges to Patient Care

10

SHERYL A. MAHONEY Indianapolis

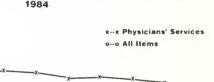
HY, SOMEONE ALWAYS seems to be asking, are health care costs forever on the rise? The following data, derived from reports of the American Medical Association, the American College of Hospital Administrators, interviews with five CEOs of Indianapolis' largest health care institutions, and 56 responses from questionnaires sent to CEOs of 114 Indiana hospitals, may shed some light on the question.

In 1980, health care expenditures in America comprised 9.2% of the GNP at \$220 billion; in 1950, \$12 billion at 4.5%. For 1985, the percentage will probably increase. The consumer price index (CPI) measured the rate of inflation in 1984 at 4.0%. Though health care costs alone are greater than the rate of inflation, health care costs are still decreasing. The 6.1% increase experienced in 1984 was the lowest in the past decade; for comparisons, please see Figures 1 and 2. The "medical care" service index comprises professional and hospital services, and health insurance. The single component, "medical care," can be evaluated bet-

ter from Figure 2, noting a reduction in overall costs of medical care the last four years, with the greatest drop in

Legislation

A total of 250 health-related bills were presented in the 1985 session of the Indiana General Assembly; 75 survived their house of origin, indicating that medical care bills are extremely important to our legislators. For example, much time and effort were devoted to controversial issues such as HEA 1075, the "Living Will" bill, allowing a terminally ill patient to refuse lifeprolonging treatment, to die gracefully if he so chooses. S.B. 510 dealt more directly with rising health care costs. However, S.B. 510, requiring the state to "study hospitals' bad debt and charity case loads" never made it out of committee.2



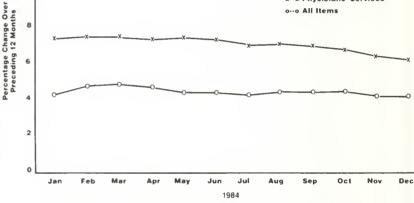


Figure 1

Twelve Month Percentage Changes in the All Items and Physicians' Service Indices of the Consumer Price Index

Source: Prepared by the Center for Health Policy Research, American Medical Association.

Based on data from the Bureau of Labor Statistics.

Health Insurance

The following question was posed to 114 of Indiana's hospital administrators: Which is most to blame for the increase in America's uninsured—unemployment or rising health care costs? The 56 respondents revealed that if health care were to cost less, most employers would provide it. That health care costs have risen has caused more people and companies to reduce, even reject, health insurance. Because health care costs have increased, benefits are cut by employers, no longer compensating their employees with attractive medical plans. The following, an exerpt from Dr. James O. Hepner's article in HealthHospital & Services Administration, supports these views:

"Of all the criticism directed against spiraling health care provider costs, the most devastating may not be from

The author is a staff member of the Indiana State Medical Association.

Acknowledgments: Lenny Emmanuel, M.B.A., Indiana University Medical Center, for editorial assistance; and Kenneth Badger and Nicky Harmon for secretarial assistance.

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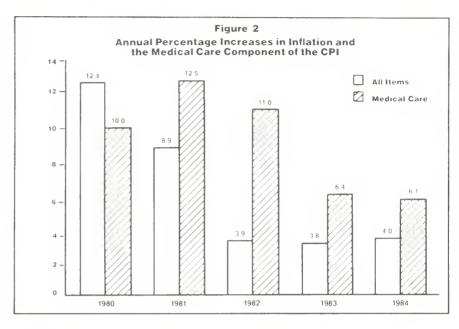
the individual consumer, but from American industry. . . . Many industries find that medical benefits are their most rapidly climbing expense . . . amounting to over \$40 billion for employee health insurance, with a comparable amount on related programs such as sick leave and disability. Costs have risen at a pace that, in recent years, has been twice the rate of general inflation. American industry now knows of 19 developed nations which spend less per person for medical care." 3

Physician Fee Freeze

The current fee freeze is again pending inclusion in the budget package. The AMA Board of Trustees has called on the nation's physicians to freeze their fees voluntarily for one year ... "to hasten the nation's economic recovery and assist individual patients who may be unable to afford care. . . .' Bob Sullivan, ISMA field representative, states "... a report by the National Center for Health Services Research indicates that about 27% of the population, or 50.7 million people, are underinsured or uninsured for all or part of the year."4 The voluntary fee freeze, according to one CEO, would have had an influence, but only by means of postponing fee increases . . . unless a shift would occur in supply and demand levels. Additional concern, involving reimbursement to the physician at rates less than costs to provide that same care as well as adding to the current problem of "cost shifting," has become alarming. Nevertheless, 80% of the nation's physicians have complied with the request of freezing fees.

Medicare and Medicaid

However, 37 organizations, including the AMA and AHA, recently signed a letter to Congress opposing proposed cuts in Medicare and Medicaid programs. The Reagan Administration has proposed cuts totaling \$24 billion for 1986-88, the letter stated. These programs, which provide vital health care to 30 million of the elderly



and 22 million of our financially disadvantaged, are once again the targets of an unfairly large burden of the spending reductions," the signatories said. Medicare and Medicaid recipients have had to absorb \$22 billion in reductions since 1981.⁵

Vic Caleca, in The Indianapolis Starb stated, "The federal government has adopted regulations that will allow the nation's 30 million elderly Medicare recipients to easily join health maintenance organizations and other prepaid health plans." A health maintenance organization (HMO) provides health care to voluntarily enroll ed individuals and families in a par ticular geographic area by member physicians with limited referral to outside specialists; members are assessed fixed periodic payments determined in advance. This announcement had distinct importance to Indianapolis HMO, Metro Health, one of the nation's select few to "test" this new concept. Under recent regulations, HMOs will receive monthly fees from the federal government for Medicare patients who choose to join such organizations. Moreover, many HMOs will make extra provisions for a nominal fee, such as for eyeglasses and prescription

drugs.

Medicare patients, nonetheless, see a definite drawback to membership in an HMO, e.g., Metro Health; it requires its members "to use only the physicians and medical facilities approved by the organization," according to The Indianapolis Star." Mr. Caleca explains that "Medicare recipients who decide to join an HMO ... will be free to cancel their membership and return to the standard Medicare program at any time. . . ." The Senate added an amendment to H.B. 1922 which would guarantee that insurance companies pay valid claims within 45 business days instead of the proposed 30 business days.

Medical Malpractice

Perhaps the most crucial variable in the rise of health care costs, however, is medical malpractice. James Sammons, M.D., AMA executive vice-president, recently said on "Good Morning America" that in 1984, one of every six physicians had a malpractice suit pending, whereas in 1985, the AMA predicts that 20% of all M.D.s will have a medical malpractice suit filed against them. Dr. Sammons also said that over 90% of malpractice suits

are without significant justification, and such cases have required physicians to acquire additional malpractice insurance, then compensate by adjusting their fees to the health care consumer." Moreover, ordering extra tests as a reinforcing protective measure has increased in popularity. These practices to help avoid litigations have increased health care costs each year by \$15 billion, Dr. Sammons said in a recent teleconference."

Although the nation is experienci. a "medical malpractice crisis," the evidence of such a crisis in Indiana is, comparatively, not as devastating. Dr. Lawrence E. Allen, M.D., president of the Indiana State Medical Association, says that "Americans are filing more than three times as many professional liability claims as they did 10 years ago and are winning record settlements."10 He says that in Indiana "our professional liability risk is comparably benign," and that "last year our premium rates averaged 2.5 to 4 times less than the surrounding states at our borders . . . which translates in lower health care costs...," an advantage made possible by Public Law 146.

Cost Shifting

Another challenge facing us is indigent care-those who are unemployed, have no medical insurance and have no means to obtain coverage. In the 1985 session of the Indiana General Assembly several bills were introduced that would have helped the "medically needy" but none was passed into law. Care for indigents has increased costs, often resulting in "cost-shifting," which occurs when one department does not produce sufficient revenue to cover the costs of a par ticular service. Funds are then required from another account to cover the expenses; this is how Ford Motors absorbed the cost of the Edsel, which was not "cost effective." The health care industry faces the same challenge, with CEOs either mustering new ideas of cost-effectiveness or implementing cost shifting and creative accounting. CEOs say that "somebody has to pay" and so cost shifting is encouraged to maintain quality patient care. One CEO said that although he does not favor cost-shifting, "without it in this day of government not paying all our costs for patient care, our hospital could not survive."

The Terminally Ill

H.E.A. 1075, another controversial issue with impact, allows terminally ill patients or their parent(s) the right to turn off artificial life. Twenty-two states have previously adopted some form of the "living will" legislation, but Indiana more recently had much disagreement among legislators, public committees, and private "movements." "Pro-lifers," as well, face the problem of preserving life at all costs. Neil Peirce, writing in The Indianapolis Star, 11 says that "28% of the country's \$75 billion yearly Medicare budget is used to maintain the elderly "mostly during the last month of their lives," and this percent "almost equals all federal aid for the nation's jobless." He also states that to "prolong the death of terminally ill patients easily costs \$20,000 to \$50,000 per case." Also, Dr. George Crile, Jr. of the Cleveland Clinic wrote recently in USA Today that "no insurance company, national or private, should be obligated to sustain the hopeless lives of those who wish to die and whose families agree."

Joint Ventures

Another issue affecting medical economics is the new role of physicians as hospital administrators, members of governing boards, and participants in joint ventures. Don Foy, executive director of the Indiana State Medical Association, made pertinent comment about joint ventures: 1) The way one's peers practice medicine makes a difference if one's money is riding on it, 2) Credentialing is underway to cull those with any business being in this kind of practice, and 3) if one wants control of the venture, then one has to own part of the venture, which means in

vesting cash and/or capital.12

To offset rising costs, therefore, joint ventures are occurring. Mr. Foy points out that although hospitals are still moving into new ventures, at least now, instead of competing with their staffs, a growing number are offering physicians a "piece of the action." He attributes this to the problems of "physician glut, proliferation of HMOs, PPOs and convenience clinics" as well as to pressure by government and business to contain costs. He notes that, on average, physicians are seeing 28 fewer patients a week than they did 10 years ago and that, in 1984, hospitals nationwide experienced a 2% drop in admissions and two days in length of stay. Joint ventures occur, Mr. Foy explains, when a hospital and one or more members of its staff go into business together, both taking financial risks in order to survive the contemporary cost-containment crisis.12

Physicians are simply becoming more cost-containment conscious than they were in the past; they are assuming increased responsibility in the administrative aspects of medicine. An Arthur Young Physician/Administrator Survey (August 1984) revealed that 70% of the respondents believed that hospital governing boards are more confident in administrators who are also physicians. According to the American College of Hospital Administrators, the number of physicians in administrative roles in 1982 and 1983 increased by 27%.13 That trend probably will continue.

Conclusion

Legislation affecting medical services, while necessarily considering cost containment, must not affect the quality of patient care. Herein lies the challenge. Doctors don't want to fall into the trap of practicing "cookbook medicine" in which patients are treated according to strict rules, rather than according to their needs. The quality of medical care cannot be restricted simply on the basis of cost considerations.

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FUTURE FILE

CONTINUED FROM PAGE 650

Practice Management

The ISMA and the Resident Medical Society will co-sponsor a practice management workshop Sept. 6 and 7 at the I.U. Medical Center.

Staff from the AMA Dept. of Practice Management will discuss patient relations, accounting systems, personnel management, third-party payers and medical records. ISMA legal counsel will review corporate and malpractice law requirements in Indiana.

Registration fees have been discounted for ISMA members, their spouses and office staff. For more information, contact Carol Ann Cunningham at ISMA headquarters, Indianapolis.

Hand Surgery

Weekend Review Courses in Hand Surgery, conducted by the American Society for Surgery of the Hand, will be held in Indianapolis Sept. 7 and 8. Tuition is \$100 for physicians, \$60 for training residents and fellows.

Inquire by writing to the society at 3025 S. Parker Road, Suite 65, Aurora, Colo. 80014.

Community Cancer Care

The Fourth National Seminar on Community Cancer Care will meet Oct. 17 to 20 at the Hyatt Regency, Indianapolis. It is sponsored by a number of Indiana hospitals and by the Association of Community Cancer Centers of Washington, D.C., and the American Cancer Society.

For a copy of the program and other details, write or phone Dixie Mattingly, 1604 N. Capitol Ave., Indianapolis 46202—(317) 929-3733. (The program is briefly outlined in *Cancer Corner*, located elsewhere in this issue.)

Echocardiography

"Advanced Echocardiography" is the theme of a CME program to be conducted by the American College of Cardiology at the Hyatt Regency, Indianapolis, Sept. 30 to Oct. 2.

Dr. Harvey Feigenbaum is the program director. The registration fee is \$315 for ACC members, \$415 for nonmembers. The course rates 16 AMA Category 1 credit hours.

For more information, contact the ACC, Extramural Programs Dept., 9111 Old Georgetown Road, Bethesda, Md. 20814—(301) 897-5400.

Cancer Conference

The Cincinnati Cancer Conference IV will meet Nov. 1 and 2 at the Hyatt Regency, Cincinnati.

Each of the three half-day programs will treat a separate subject—Ovarian Cancer, New Developments in Cancer, and Melanoma. Nursing issues will be discussed at a separate meeting for nurses at the same location and times.

For a copy of the program and a registration form, write to CME, Bethesda Hospital, Location 00348, Cincinnati, Ohio 45264.

Cardiology Update

"Update in Cardiology: Cardiovascular Board Review" is the title of a four-day CME program to be conducted by the American College of Cardiology at the Hyatt Regency, Indianapolis, Sept. 5-8.

The program directors will be Dr. R. Joe Noble and Dr. Charles Fisch, both of Indianapolis. The registration fee is \$475 for ACC members, \$525 for non-members. The program is approved for 36 AMA Category 1 credit hours.

Contact ACC, Extramural Programs Dept., 9111 Old Georgetown Road Bethesda, Md. 20814—(301) 897-5400, ext. 230.

Medical Malpractice—1985

Reflections of a Health Care Provider

M. MARTIN HALLEY, M.D., J.D. Topeka, Kansas

■HE MALPRACTICE PROBLEM is again approaching crisis proportions, impacting upon patients, competent providers, law, the insurance industry, the economy, government, and society. Primary factors, frequently obscured, are patient injuries, a by-product of modern health care, sometimes resulting from providers' negligence. The analogy to industrial injuries is apparent. The tort system, based on "fault," continues to be ineffective for reasonable and prompt compensation. The same system was condemned as anti-social and oppressive during the development of workers' compensation programs.

Innovative concepts increasingly recognize the health care injury, and compensation and assistance in rehabilitation without the proof of fault. Model programs are available in the New Zealand Accident Compensa-

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Reprinted with permission from *The Jour*nal of the Kansas Medical Society, December 1984. tion Act, The Swedish Patient Injury Insurance Plan, and Workers' Compensation in the United States. The ultimate solution is replacement of our fault and tort law structure with an innovative no fault compensation system.

The problem of medical malpractice, more accurately referred to as medical professional liability, remains unresolved. Health care delivery crises again appear imminent, signaled by increasing claim frequency, rising claim severity, and escalating insurance premiums. Basic issues involved in the continuing controversy are generally not well understood, or may be distorted by the intensity or selfinterest of the parties, creating difficulty in objective evaluation. Adequate data are not easily available, so that analysis producing meaningful information for decision making is difficult.1-5

What is the medical malpractice problem? Where does it impact? How can it be solved? These questions will be addressed in this discussion in terms of past and present developments in order to identify a potential long-term solution.

The Problem

In 1971, the pervasive nature of the malpractice problem focused national attention on the subject, not due merely to the rising volume of malpractice claims, but due to concern for their potential impact on the entire health care system. A presidential directive convened a commission on malpractice, and included the following observation: "The consequences of the malpractice problem are profound. It must be con-

fronted soon, and it must be confronted effectively, but that will be no simple matter. For one thing, we need to know far more than we presently do about the complex problem...."⁵

Today we do know more about the problem, but reasonable minds still differ on its precise definition. It is not primarily a problem of substandard health care practices, solvable through risk management or disciplinary action against providers. It is not primarily a controversy between physicians and trial attorneys, or between health care providers generally and the legal profession, although reports and news media coverage of events may convey this interpretation. It is not primarily a problem of the insurance industry, manifested by rising premiums, although this sector is certainly involved, as are the economy, state legislatures, state regulatory agencies, and the federal government. It is primarily, however, a problem of patient injuries, real or imagined, arising out of or in the course of health care delivery, and at times resulting from health care providers' negligence.5 It is a problem of personal injury to patients in the environment of high technology-modern health care, multiple treatment modalities and drugs, an astronomical number of decisions or individual judgments for delivery or non-delivery of care, and the occurrence of suboptimal or bad results or treatment failures, sometimes in patients who formerly might not have survived.

The problem is ultimately one of society, wherein the analogy of industrial injuries to health care injuries is increasingly apparent. The former presented as a by-product of industrial-

ization, and because of public concern for workers and their families, resulted in legislation for the protection of workers against the special hazards intrinsic in an industrial society, first in Europe, and subsequently in the United States.6 The latter are an increasing hazard to patients, as an unfortunate by-product of modern health care. Societal concern should. therefore, result in a solution providing protection and compensation - reasonable and expeditious and without the uncertainties related to determination of fault-for patients against the special risks intrinsic in health care delivery.

Historical Perspective

The Code of Hammurabi in 2250 B.C. prescribed penalties for physicians who caused loss of life, or loss of an eye. Several English malpractice cases ap peared in the 1700s, and the first United States case was reported in 1794. The incidence of claims was then relatively insignificant for nearly 140 years, but in the decade 1930-1940, the number of claims rose tenfold. Another tenfold increase occurred during the decade 1940-1950, and since 1950 the trend in claim frequency and severity has continued.7 In 1975, a national crisis in insurance availability and cost resulted in various legislative remedies involving tort law reform, frequently combined with disciplinary measures for health care providers. Liability insurance thereafter again became available. Claim frequency, severity, and insurance costs are presently escalating, and settlements, awards, or judgments in seven figures are increasingly common.

Paradoxically, the phenomenon of increasing malpractice claims and awards is occurring in a society where health care practice and achievement have attained heights previously unimaginable, and where great scientific and technological advances continue. The phenomenon is not limited to health care, but is one segment of personal injury litigation prevalent in

the United States today, which includes automobile liability, product liability, air and rail liability, home owner liability, as well as professional liability generally. Medical malpractice is, therefore, a part of a general trend in tort litigation, although a number of more specific causes are as follows: diminished rapport with patients accompanying the technological and medical advances: unrealistic consumer expectations and consumer frustrations; an increasingly litigious society; an increasing number of highly skilled and increasingly specialized attorneys working in the context of the contingent fee; increasing emphasis in law school curricula upon medical malpractice; news media publicity for all kinds of medical affairs; and the influence of increasingly large awards, judgments,

and settlements. Pro-plaintiff changes in law in recent years have also been significant. These changes include abolishment of the doctrines of charitable and governmental immunity for institutions; expansion of the locality rule for medical standards and the rules for expert witness qualification; findings of oral guarantees; long statutes of limitation; liberal application of the discovery rule; application or extension of the doctrines of res ipsa loquitur and informed consent; liberalization of doctrines relating to prenatal or perinatal injury; and extension of concepts of mental suffering or emotional disturbance. Finally, proplaintiff changes include court expansion of tort law doctrines into strict liability to compensate an injured plaintiff in the absence of demonstrable fault, where the defendant, through the use of insurance, is the more responsible person. The courts are hereby compensating individuals who suffer damages through no fault of their own by assessing damages against health care providers. Thus they "spread the risk," predicating compensation not upon the liability of an individual defendant, but upon the existence of a "deep pocket" or an insurance fund able to pay the compensation.8,9

Basis of Liability

Liability in the present tort system is frequently based on negligence although other legal theories may be applied. Negligence actions involve (1) a duty as arising out of the physicianpatient relationship; (2) breach of this duty by deviation from the standard of care; (3) damage to the plaintiff; and (4) a causal relationship between the breach of duty and the damages. The legal concept of the standard is stated in court decisions as the duty of the physician to possess and exercise that degree of care and skill that is expected of a reasonably competent practitioner in the same class, acting in the same or similar circumstances.10

The standard of care may be visualized graphically as the density function of a continuous random variable, the bell-shaped curve, a probability distribution applicable to biologic variables such as cognition, decisions, and actions. Sixty-eight per cent of behavior will fall within one standard deviation of the mean, 95 per cent within two standard deviations, and 99.7 per cent within three standard deviations. Assuming the separation of relatively good and relatively bad practice to occur at the mean, a predictable percentage of actions or decisions will be good, better, bad, or worse. Thirtyfour per cent will occur within one standard deviation, 13.7 per cent will occur within one and two standard deviations, and 2.3 per cent will occur within two and three standard deviations on either side of the mean. It follows that a practitioner, no matter how knowledgeable, how competent, or how skillful, will make sub-standard decisions or perform sub-standard acts on a statistically predictable basis. The performance curve may additionally be shifted unfavorably by other circumstances such as relative cognitive ability, decisions or actions outside the practitioner's major expertise, fatigue, distraction, over-extension, behavior of assistants or others, as well as by technical, environmental, or patient factors. Even if all decisions or actions

are acceptable, a number of bad results will be similarly predictable, and "fault" may be found through application of strict liability. The bell-shaped curve thus illustrates a major defect in the tort system, presenting competent practitioners with unfavorable probabilities or "negligence" and, on the other hand, requiring injured patients to prove the deviation from the mean and "fault." The combined effects of this and other defects of the tort system can be summarized as follows: There is no objective standard of liability; there is no definite measure of compensation; the entire process is susceptible to subjective considerations; the cost of litigation is high, in expenses and attorneys' fees; there is no restraint mechanism to litigation; there is no encouragement for prompt settlement; and finally, the system encourages and facilitates ever increasing awards.

The same traditional tort system applicable to industrial injuries prior to the enactment of workers' compensation statutes was condemned as antisocial and oppressive in 1909-1910 reports to the State of New York legislature. Investigating commissions unanimously concluded that (a) a large portion of all fatal and non-fatal injuries remained uncompensated, (b) the sums actually paid were frequently inadequate token compensation, (c) recoveries were obtained only after protracted litigation, (d) the attorneys of the injured workers retained a large share of the sum actually obtained, and (e) an undue portion of the premiums paid by industry went to insurance companies for profits and administrative costs and was thus socially wasted.6

Impact: Health Care Consumer

Patient injuries, real or imagined, are prime factors in the malpractice problem,⁵ which is additionally affected by other causes. A number of generally beneficial recommendations have been made intended to minimize such injuries, but none can be expected to

significantly abate the problem. There is no evidence that more or better education in our already lengthy health care programs, expanded disciplinary procedures against health care providers, more or better post-graduate education. increased emphasis on hospital licensing, hospital staff regulation, or other measures to encourage professional competence would have a significant impact.

In the patient's view when all is said and done, health care is a necessity and a right, but includes an inherent risk of injury. Compensation and rehabilitation must, therefore, be emphasized, but both are slow and uncertain under the present tort system. Injured patients, when compensation occurs, ultimately receive 20 per cent or less of the insurance dollar. The overall effect appears to be a wind-fall for a few patients, large rewards for a few attorneys, and income for insurance companies, defense attorneys, and others involved in the system.

Impact: Health Care Providers

The impact on health care is not only a matter of financial burden to providers, or increased cost to the public which ultimately pays both the direct costs of insurance and the indirect costs of defensive practices. The problem has been noted to touch every facet of our health care delivery system, including costs, patterns of medical practice, forms of treatment, the distribution of health manpower, the relationships between physicians and patients, and even confidence in equal justice before the law.⁵

Undesirable effects occur in the physician-patient relationship, since physicians must increasingly view each patient as a potential plaintiff. Widespread defensive practices incur additional inconvenience, cost, and risk to patients in the form of longer or earlier hospitalizations, an increased number of procedures or tests, recommendations against some procedures for legal rather than medical reasons, more consultations, early referrals to other

physicians, stricter limitation of practice, withdrawal from emergency service, or early retirement of experienced physicians. Another significant problem may be physician dysfunction subsequent to the filing of a claim, or during trial. On the other hand, beneficial effects of defensive practices have been noted, since these may also be good patient care, and quality control benefits for health care through the threat of tort litigation have been suggested.

Yet another area of impact is the increasing participation by physicians or other health care providers in claim review for attorneys or as medical witnesses. A recent estimate based on analysis of national advertising material indicates that 3,000 physicians are active in this process. Remuneration is substantial: \$500 for chart review, analysis or report; \$600 for depositions; and \$3,000-\$5,000 daily, plus expenses for court appearances. Thus the alleged medical "conspiracy of silence" - once a chronic complaint of the legal profession concerning difficulty in obtaining medical witnesses has been replaced by vigorous marketplace activity, with numerous physicians competing for the opportunity to participate in a lucrative field. Availability of abundant medical testimony and assistance in case preparation is believed to be one of the catalysts that has opened the floodgate of professional liability litigation in the medical field.11

Impact: The Legal System

The legal system, consisting of attorneys, courts and law, has been a fundamental factor in the present malpractice problem, most importantly through the expansive application of the fault and liability concepts of tort law. Attorneys and judiciary will continue to be important in the developing efforts to restructure the tort system, and can be expected to resist change in terms of fault, liability, adversary and litigation, concepts deeply ingrained in Anglo-American

law. Law students and attorneys are firmly attached to the adversarial process which requires parties to "battle" to reach truth and justice, a process that essentially renders only a victor. Law students and attorneys are less familiar with the terms negotiation, settlements, mediation, and compensation without fault. Therefore, the "fault" frame of reference continues to be the legal profession's response to many societal problems, as contrasted to the "compensation" system based on a foundation other than "fault." 12

Certain other factors involving the legal system deserve mention. The increasing number of attorneys - which has resulted in manpower for specialization and increasing expertise - has been suggested as a major factor in the escalation of the malpractice problem. Attorneys-both plaintiffs and defense - with special interest in this field, contribute not only time and skill to legal issues, but exercise considerable legislative influence to actively or passively oppose changes in a framework that has identified health care with other major target defendants. The legal system thus professes to protect the rights of patients and strives for injury compensation within the present tort law, but these objectives are at times obscured by seemingly inappropriate tort law results, as well as by highly visible controversies involving opposition to legislative reforms of tort law structure.

Impact: Insurance

The insurance industry at the time of the 1975 crisis was in a state of near collapse, manifested by carrier withdrawal from the marketplace and by increasing premiums. State legislatures then enacted a variety of tort law modifications, and frequency of claims declined for three years, but claim severity continued to increase. In the 17 states where a financial limitation was placed on awards, and in the 16 states where the collateral source rule was repealed or modified, claim severity was reduced, but no effect on fre-

quency was noted. No other substantive legal reform was found to have statistical significance in reducing either the frequency or the severity of claims.¹³

In Kansas, the Health Care Stabilization Fund, established in 1976 under the Health Care Providers' Insurance Availability Act, was intended to be accompanied by a limitation on total awards. 14,15 The limitation, however, was never seriously considered by the tegislature, and by 1983 the fund was actuarially insolvent. Legislative intervention was required: Basic insurance coverage requirement was increased, excess coverage available from the fund was limited, and the levy of the fund surcharge was changed to an accrual basis. As a result, health eare providers' insurance premiums increased substantially.

The Kansas Insurance Department reported on June 30, 1984, that awards against the fund totaled \$22,222,605. Ninety-five awards had been paid with an average payment of \$233,912. A total of 710 cases had been filed, and 365 remained active. Most significantly, there had been eight claim awards, judgments, or settlements in the million-dollar range in the past 15 months. The increasing exposure of the fund, together with a continuing unfunded liability, may necessitate additional corrective legislation, as well as studies of insurance alternatives to its continuation

Impact: Economy

The economic impact of malpractice is difficult to measure, although it is generally believed to be substantial. Present emphasis on health care cost-control focuses attention on this aspect of an industry accounting for 10.9 per cent of the gross national product. A 1983 report estimated direct annual malpractice costs—the insurance premiums paid by physicians—as \$1.75 billion, but could not estimate hospital premium costs, which included liability premiums, risk management programs, and other miscellaneous items.

Indirect costs, attributed to defensive medical practices, were estimated as \$15.1 billion, but a number of these practices may not be strictly defensive. Current direct costs are reported as \$3.5 billion, and indirect cost estimates range up to 30 per cent of total health care expenditures.

Impact: Government

State legislatures have been, and continue to be, the arena where reforms must be obtained, but remedies have been variable and have not resulted in a long-term solution. In Kansas and other states, legislation has produced only "piecemeal" reforms that have principally resulted in continuing availability of liability insurance.

The current legislative program of the Kansas Medical Society again consists of tort reform proposals. It will include proposals for limitation of total awards, limitation of pain and suffering, collateral source revision, modification of attorney's fees, itemization requirements for verdicts, reduction of the judgment interest rate from the eurrent 15 per cent, allowing periodic payments to expire at the death of a plaintiff, and a procedure providing incentive to early settlement. Legislative consideration of these proposals will require concerted action by health care providers, including coalitions with other interests affected by present tort liability problems. Other requirements are general agreement on the program and its objectives, adequate funding, news media exposure, public education, legislative information programs, individual contacts with legislators, and perhaps a governor's task force to address important issues.

At the federal level, interest in medical malpractice is inevitable, since the government is the largest single purchaser of health care services. In the past, this interest has been passive, but in April 1984, an "Alternative Medical Liability Act" was introduced in the House by Representatives Moore and Gephart, incorporating previously published recommenda-

tions, 16 and was referred to the Committee on Ways and Means. This bill provided for settlement of malpractice claims arising in programs established under federal law. The central feature permitted a health care provider potentially liable for personal injury to tender compensation for the claimant's net economic loss, and by this act, to foreclose tort law litigation.

Medical opposition to the bill focused on its doubtful effect on defensive medical practices, the possible cost impact of an increased number of claims, problems of court involvement, and issues related to provider decisions, third party joinders, and federal intervention. The appearance of these concepts at federal level may signal a more active posture, and may serve to encourage state solutions in the hope of avoiding further federal action.

Discussion

Efforts to resolve the malpractice problem in the United States have generally involved insurance alternatives or tort law modification, as well as recommendations for preventive action in the health eare industry. Insurance programs are of obvious importance, since availability and reasonable premium structure are essential to continuing health care practice. Tort law modification has been difficult to obtain, and has not proven significantly effective, since available data indicate that only total award limitation and collateral source modification have produced measur able short term mitigation. Preventive programs are important, but an ir reducible number of injuries will never theless occur.

Innovative concepts are, therefore, assuming increasing significance. Their overall thrust is the evolving recognition of the health care injury arising out of modern health care delivery, and the increasing consensus that compensation for such injury, and assistance in rehabilitation, should not depend upon the proof of fault. The Definition of the injury, or compensable event, con

tinues to be the major challenge in several available studies. 22,23 Initial concerns about unfavorable cost impact are being re-evaluated in the light of present monetary values and enormous awards. Other components of a compensation system include the measurement of damages, the form or amount of compensation, the source of compensation funds, case-disposition mechanisms, fund collection and disbursement, and methods of dispute resolution. Three apparently successful injury compensation systems, the New Zealand Accident Compensation Act,24 27 the Swedish Patient Injury Insurance Plan.28 30 and the Workers' Compensation System in the United States, 6,31 are available models for analysis and comparison.

New Zealand, since 1974, has defined personal injury by accident to include "medical, surgical, dental, or first aid misadventure." The program merges workers' compensation and automobile protection, and adds coverage for victims of other accidents. Common law actions for negligence are precluded to the extent that an injury is compensable under the no fault system, but it is not yet clear which cases will be compensated and which will be litigated. Financing is by levies on employers and self-employed persons, levies on owners and drivers of motor vehicles, and money appropriated by parliament.

Benefits include medical care, transportation to the physician or hospital, funeral expenses, awards for permanent loss or impairment of function, payment for lost earnings, and timited awards for disfigurement or pain and suffering. In exchange for the new program, the injured person has traded the dubious advantage of litigation of torts for a quick and informal administrative procedure. He has traded the possibility of a large award for a more certain, modest payment for injuries, limited pain and suffering, and assurances of income maintenance.

After four years of operation, this innovative program appeared to be

working well. Claims were processed rapidly and routinely, and with an acceptable administrative cost. Few claims were appealed beyond the initial level and most were paid without at torney involvement. One of the major difficulties was ascertaining the range of coverage intended by the statutory words.

Sweden, in 1975, established a patient insurance program after general realization that few medical malpractice claims resulted in compensation for the patient. The primary goal was to create a provider-financed scheme for compensating victims of significent medical injuries in three categories; (1) an injury that occurred as a direct consequence of examination, medication, and treatment, and that was not associated with known risks, but excluding injuries or illnesses likely to have arisen irrespective of care rendered; (2) an injury that occurred as a result of incorrect diagnosis or an incorrect interpretation of symptoms, that is, procedures not reflecting generally accepted medical practice; and (3) an injury that occurred as a result of a sudden external event within the health care institution or during transportation by ambulance.

Negligence was discarded as a standard of payment, and the mechanism for discipline of providers was separated from the compensation program. Insurance coverage to pay claimants is purchased from a pool of private carriers by private and government-employed health care providers. Claims are submitted to an insurance office, and most are then paid directly. Appeals are possible to a claims panel, and thereafter may be submitted to arbitration. The patient initially retains the right to proceed in the court system, or may file a complaint with the National Board of Health and Welfare.

Objectives of the program were to overcome some of the disadvantages in the prior fault based system, and to accomplish speedy resolution of claims without the adversary process and litigation. Results of the program do not indicate generation of unduly large numbers of claims, and do not indicate extensive investigation of claims or contests in the level of awards. Review of the program's development will establish the extent of these results, as well as the effects of separating the compensatory side of medical malpractice from the disciplinary aspects.

Workers' Compensation, the oldest branch of modern social insurance, became part of the European legal system long before its acceptance in the United States. Beginning in Germany with the enactment of an Employer's Liability Law in 1871, a number of other continental powers adopted industrial accident insurance acts before the turn of the century. England followed with the Employer's Liability Act of 1880. In the United States, in 1909, Montana enacted a state compensation system for the coal mining industry. Subsequently in 1911, the largest number of state statutes were enacted, and in 1917 constitutional barriers were removed when the Federal Supreme Court upheld the three existing types of compensation laws. The adoption of a compensation act by Mississippi in 1948 made the system universal.

Thus, in a span of approximately 80 years, the inadequacies of the tort system were gradually corrected so that the victims of industrial accidents and their families were adequately and promptly protected. New legal principles were needed, but legislators were slow to grasp this necessity. These new principles established that the great bulk of work accidents should be regarded as part of the unavoidable loss of modern industrial operations, and should not be approached with concepts of fault. The accident toll present ly in American industry is nearly 43 million working days annually; at least 16,500 deaths occur each year through routine industrial operations; and accidental limitation of earning capacity involves more than 2 million other workers. Compensation is payable according to a definite scheme. Payment is secured by employers through private insurance, state funds, or self insurance. Fault has been eliminated. The compensation represents a compromise in which each party surrenders certain advantages in order to gain others more important to him/her and to society. Employers give up the immunity they would enjoy in cases where they are not at fault, and employees accept a smaller, but certain and prompt compensation.

This system appears to have resolved the problems of industrial accident compensation, and no serious argument has appeared for return to the tort system. Should it not be asked why the same scheme is not equally appropriate for many other injuries presently administered under the traditional fault system of tort law?

Conclusions

Tort law approaches to the medical malpractice problem have not resulted in a permanent solution due to the inherent disadvantages of the fault ap proach. In health care, profoundly negative effects involve both delivery and cost. In law, pro-plaintiff changes have further extended tort law application into the realm of strict liability, and segments of the legal system have effectively opposed most legislative reforms. In government, legislation to date has provided only short-term relief, has been accompanied by disciplinary measures for providers, and has principally resulted in availability of insurance to pay the ever increasing awards and settlements.

For the patient, high quality health care has been accepted as an individual right in modern society, accompanied by the growing awareness that such care includes inherent risks of personal injury related to the health care process, rather than to the underlying ill ness. For society, the concept of the health care injury, strikingly similar to the industrial accident, appears in creasingly attractive as a long-term solution of this complex problem.

awarding compensation based on no fault principles.

Many parallels exist in the evolution of the malpractice problem and in the development of workers' compensation systems, as well as in the development of present no fault compensation programs for health care injuries in New Zealand and in Sweden. The ultimate solution is thus on the horizon, as the replacement of our venerable fault and tort law structure by an innovative no fault compensation system.

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AUXOLOARY REPORT

Muriel Osborne (Mrs. John) ISMA Auxiliary President 1985-86

Indiana delegates to the American Medical Association Auxiliary 1985 annual convention gathered in Chicago on Saturday, June 16. Several of the delegates dined in the Sears Tower on Saturday evening with the Indiana delegation to the AMA convention, being held at the Marriott Hotel in Chicago.

The AMA Auxiliary convention began Sunday morning with National Previews at which Mary Kay McPhee, president-elect, presided. Briefing sessions were held on the Auxiliary's four areas of concern: Membership, AMA-ERF, Health Projects, and Legislation.

Sunday evening the formal opening of the 62nd annual session of the AMA Auxiliary was held in the Grand Ballroom of the Drake Hotel. The keynote address, Health Care in America, was given by the Honorable David Durenburger, Republican Senator from Minnesota. Senator Durenburger complimented the Auxiliary on their positive influence on health care in America. A reception in the Gold Coast Room in the Drake Hotel honored President Billie Brady and President-elect Mary Kay McPhee.

ISMA Auxiliary delegates attended the AMPAC breakfast Monday, June 17, and were told by Edie Epstein, Auxiliary member of the AMPAC board of directors, of the need for auxilians to join AMPAC. The breakfast was followed by reference committee hearings.

On Wednesday the convention approved the Health Issues Reference Committee recommendations that county and state auxiliaries: support programs to educate the public about the importance of early detection of colorectal cancer; establish programs for spouses of impaired physicians; promote educational programs to encourage the use of protective helmets



OUR DELEGATES—Seated, from left: Suzanne Miller. Anne Throop, Muriel Osborne, Alfrieda Mackel, Dorothy Bickers and Lura Stone. Standing, from left: Marge Smith, Wilma Jean Scamahorn, Judy Koontz, Rosanna Iler (ISMA Auxiliary Liaison), Martha Stout, Lucreta Allen and Carole Wainscott. Not shown: Barbara Lukemeyer and Vivian Priddy.

to prevent serious head injuries associated with off road accidents; and support programs to educate the public about anorexia nervosa and bulimia. The above resolutions were to be taken with approval of and in cooperation with corresponding state and county medical societies. At a luncheon honoring national past presidents, Mark Russell, political humorist, entertained delegates with comments and songs about current political personalities and events. Uwe E. Reinhardt, Ph.D., professor of economics, Princeton University, spoke on the Health Care Brawl at the Tuesday luncheon.

A symposium Tuesday afternoon was held by Florence Littauer on How to Understand Others by Understanding Yourself. Joseph F. Boyle, M.D., president of the American Medical Association, addressed the convention and stressed the positive role of Auxiliary in the area of organized medicine. Wednesday morning Mary Kay McPhee was installed as the 1985-86 president of the AMA Auxiliary. A buf-

fet luncheon honoring her and the 1985-86 hoard of directors was sponsored by the Missouri State Medical Association Auxiliary.

Several members of the ISMA Auxiliary delegation stayed to attend the inauguration of Harrison L. Rodgers, Jr., M.D. as president of the American Medical Association and the reception that honored Dr. Rodgers and Mrs. McPhee. ISMA Auxiliary delegates attending the convention were President Muriel Osborne (Mrs. John): Carole Wainscott (Mrs. Clinton); Judy Koontz (Mrs. James); Martha Stout (Mrs. Francis); Anne Throop (Mrs. Frank); Suzanne Miller (Mrs. John D.); Alfrieda Mackel (Mrs. Frederick); Lura Stone (Mrs. Robert); and Marge Smith (Mrs. Philip). Alternate delegates were: Vivian Priddy (Mrs. Marvin); Barbara Lukemever (Mrs. George); Lucreta Allen (Mrs. Lawrence); Wilma Jean Scamahorn (Mrs. Malcolm). ISMA liaison Rosanna Iler attended the convention with the ISMA Auxiliary delegation.

BOOK REVIEWS

A Thinker's Guide to Ultrasonic Imaging

By Raymond L. Powis, Ph.D., and Wendy J. Powis, D.M.U. Copyright 1984, Urban & Schwarzenberg, Baltimore, 417 pages, hardcorer, \$47.50

A Thinker's Guide to Ultrasonic Imaging is a fairly comprehensive text on the physical and electronic events underlying diagnostic ultrasound. The book begins with a simple explanation of wave properties and then builds on this. Each chapter, however, addresses a separate area of interest and can be read alone. Included are discussions of both B-scanning and real-time ultrasound and a chapter on the physics of Doppler. The complete ultrasound image chain from transducer to image production is discussed in detail with fairly simple explanations and drawings. There is some discussion of instrumentation, although limited.

The preface indicates that the book is directed toward "every individual who holds a transducer in hand." Although quite well written, it probably does not meet this goal. The text is well suited for an ultrasound technologist. It may also be of limited value to a radiologist with a specific in terest in ultrasound physics or one wishing discussion of basic ultrasonic principles. Otherwise, its value to a elinical radiologist is limited.—Jack J. Moss, M.D., Indianapolis, Diagnostic Radiology



Vital Signs

A novel by Barbara Wood, Copyright 1985, Doubleday and Co., Inc., Garden City, N.Y. 326 pages, hardcover, \$16.95.

There appears to be a whole genre of imaginative literature concerning doctors and medicine. The hospital milieu contains an unending source of human drama and the author here has woven the lives of three female medicos from their first day of medical school in California to mid-life (the last part concerns 1985 and 1986 – a bit of authorial clairvoyance).

This is the story of Ruth, a gynecologist, and Sondra, a G.P. practicing eventually in Kenya, and finally of Mickey, a plastic surgeon in Hawaii and southern California. In the early chapters the plot is encumbered by the three separate lives, and efforts to connect them, aside from calling them friends, seem a little contrived. As the tales of the three lady doctors unfold. the plot gets stronger. I especially was interested in Ruth who is depicted as an overachieving Jewish gal who is obsessed with her father. When he dies. Ruth has a breakdown. Her humiliated and ignored husband Arnie is far and away the most believable character. Maybe the author should have stuck with Ruth and Arnie and followed their lives in depth?

Well written eliches abound in this book. Must I say more?—Rodney A. Mannion, M.D., LaPorte, Urological Surgery

Antimicrobial Prescribing

2nd edition, by Burt R. Meyers, M.D. Copyright 1983, Antimicrobial Prescribing, Inc., Princeton, N.J. 176 pages, softcover

This graphic and concise treatise on the use of antibiotics is well written and contains a mint of information. The author first reviews the mechanism of action of antibiotics. The advantages and disadvantages, and the use of each group of antibiotics are discussed. Then, there are several paragraphs devoted to the usual treatment of infections in various organs of the body. He reviews dosages of antibiotics in patients with renal failure, the effects of dialysis on antibiotic levels, and the prophylaetic use of antibiotics.

This concise 146-page summary covers most of what is known of antibiotic therapy at this time. It is a small book, and could be an invaluable reference to practically all practitioners. The major problem with this volume is one noted by the author that one should always review the inserts with the medication defining new uses for a particular antibiotic. As with all similar type publications, there are new developments which make a few of the recommendations out of date almost by the day the book is published. It is easy to get the desired information quickly and would be in most instances, a more appropriate reference even than the PDR.-I. E. Michael, M.D., Indianapolis, Internal Medicine

Clinical Interpretation of Laboratory Tests

By Frances K Widman, M.D., Associate Professor of Pathology, Duke University School of Medicine, and Assistant Chief, Laboratory Science, VA Medical Center, Durham, N.C. 9th edition, Copyright 1983, F. A. Davis Co., Philadelphia, 602 pages, softcover, \$18,95.

In this era of rapidly expanding laboratory technology some text of this kind is required by every user of clinical laboratory tests. As the author notes in the preface, "The book is intended for the clinician responsible for whole patient care, not for the specialist who concentrates on a single aspect of disordered function."

Presumably, the elinician will use the volume 1) in interpreting results returned to him by the hospital laboratory, 2) in finding out what are the ranges of normal values in the various tests described, and 3) in planning what tests to ask for in a given clinical situation to get maximum information with the least strain on the patient and his pocketbook.

The easily found tables listed in the front of the book will supply the laboratory values. The short accounts of pathophysiology which appear under each chapter heading will be very helpful in test interpretation and selection of tests.

The first five chapters deal with disorders of blood cells and hemostasis. They are followed by chapters in immunology, body chemistry, endocrine disorders, pregnancy, urinalysis, cerebrospinal fluid, feces, sputum, gastrie and duodenal contents. There are no bibliographic references. The index at the back of the book is quite adequate.

In my opinion this book, because of its good organization and conciseness, will be very useful to busy practitioners.—Paul S. Rhoads, M.D., Richmond, Internal Medicine

Current Medical Diagnosis and Treatment 1985

Edited by Marcus A. Krupp, M.D., et al. Copyright 1985, Lange Medical Publications, Los Altos, Calif. 1,157 pages, softcover, \$27.50.

Here is the best book bargain of which this reviewer has any knowledge. It is, indeed, the medical student's reliable and economical friend and equally useful for the practicing physician. The 37 co-authors have been recruited chiefly from California medical schools. As is done each year, the text has been updated. Significant innovations and changes have been made. New and approved

drugs are included with the authors' views on their use. New radiologic techniques such as nuclear magnetic resonance imaging are discussed. New light on the mechanisms of action of immunosuppressive agents is given. How to use newer antibiotic and new drugs for parasitic diseases and new information on HLA in disease are presented along with other updated material.

The book is not to be regarded as merely a handbook for quick reference. It supplies information which is as complete as that of any single volume on medical diagnosis and treatment now available. In the preface it is stated that since 1962, when this yearly text was introduced, 23 annual revisions have been made, and many printings in foreign languages such as Italian. Greek, Spanish, German, and Turkish have been printed. Over 1.3 million volumes of the book have been purchased. Presumably, this is why the Lange texts can be produced year after year at such a modest cost.-Paul S. Rhoads, M.D., Richmond, Internal Medicine

General Urology

11th edition, edited by Donald R. Smith, M.D. Copyright 1984, Lange Medical Publications, Los Altos, Calif. 661 pages, softcover, \$24.

I first reviewed the 7th editions of this volume in 1972, then the 9th in 1978 and finally this, the 11th. *General Urology* first appeared in 1957 and was the exclusive work of Dr. Smith while now there are 33 contributors. As our

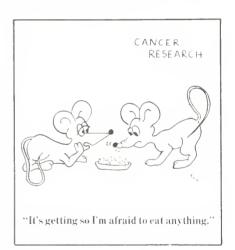
knowledge has burgeoned so has the book, which is now up to 661 pages.

For a single volume it is mightily comprehensive. This is due to the soft cover format with rather tightly spaced text and illustrations. All is of a high quality. In fact it is, in this sense, a very typical Lange scientific publication. They cover more information in a limited space than any publisher that I know.

It has been brought up to date with sections on Interventional Uroradiology, Immunology of Genitourinary Tumors, Urodynamic Studies, Clinical Andrology, and so on.

The other basic chapters on anatomy, embryology, physical examination and basic clinical urological knowledge remain and are excellent.

As always, this book is a useful reference work and recommended to any interested reader, both those still in hospital training and those in practice.—Rodney A. Mannion, M.D., LaPorte, Urological Surgery



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BOOK REVIEWS.

Swenson's Pediatric Surgery

Edited by John G. Raffensperger, M.D. Copyright 1984, Appleton-Century-Crofts, New York, N.Y. 957 pages, hard-cover, \$78.95

This is the fourth edition of what has been a standard text since the first edition in 1958. The editor is head of the division of general surgery of the Children's Memorial Hospital, Chicago, and Professor of Surgery, Northwestern University Medical School. He has enlisted a group of 38 consultants to present the various special problems encountered in pediatric surgery. The majority are colleagues with whom he works day by day in the Chicago area. Many of them are not surgeons. They represent a wide range of specialties in pediatrics.

In the introduction the editor gives his ideas on how the pediatric surgeon should view his responsibilities. Team work between surgeon, anesthesiologist, house staff, non-surgical pediatricians, nurses and other paramedical people is essential. The operating surgeon should be in charge of the whole undertaking, a responsibility that cannot be sidestepped. This requires that he must carefully review the history and bring out any additions that are pertinent. He must listen to both the parents and the young patient himself if the latter can communicate with him. To do a satisfactory physical examination is essential. This requires that he be patient and gain the cooperation of the child, who is going through a new and frightening experience. Finally, both parents and child must be told what is planned in both surgery and post-operative care and what their role in the latter is to be. Those acquainted with Dr. Raffensperger know that he practices what he preaches. In other words, he strives to be as nearly a complete physician as it is possible to be. He seems to have impressed this upon the experts who have collaborated with him in preparing this

The text is divided into 15 sections.

among which there is adequate discussion of most every surgical problem met with in children. These divisions have as their headings such titles as Preoperative Assessment and General Considerations in Care, Trauma, Gastrointestinal Hemorrhage, Anomolies of the G.I. Tract, Jaundice in Infancy, Respiratory Distress, Endocrine Disorders, and the like. Each of these is divided into up to 20 subdivisions, each presented by an expert in the area being discussed.

While the book is not primarily a text on operative technique, full descriptions of what is to be undertaken by the surgeon are given along with numerous photographs. The many hand drawings of the steps in the various operations and procedures are excellent. The discussion in each of the categories in a given field are presented by experts in those areas.

As an example of the fact that many of the disorders appearing in early childhood are, indeed, different from those in adults and must be dealt with differently, one might consider the section of the book on tumors. One learns that cancer is second only to trauma as a leading cause of death in children. Wilms tumors, neuroblastomas and tumors of the liver appear to be particularly common in early childhood. However, the majority of neoplasms seen in adults may at times afflict children also. Practically every solid tumor of the kidneys in this age group is apt to be considered a Wilms tumor till proven otherwise. Unfortunately, this neoplasm very frequently is first recognized when it presents as a large mass in the abdomen or flank. At this stage it may have spread widely to contiguous structures and even to more remote structures. If the bulk of the tumor mass can be removed surgically, follow-up radiation and chemotherapy may result in a favorable response.

Neuroblastomas and especially ganglioneuromas, while regarded as malignant neoplasms, occasionally regress spontaneously. Neuroblastomas are found usually in the retroperitoneal space but may develop from the embrymic neural crest anywhere from the base of the skull to the presacral area. The adrenal medulla arises from these same parent cells. That is why neuroblastomas often manifest the same biochemical abnormalities as adrenal tumors. However, the elevated catecholamines found in the former seldom produce symptoms beyond diarrhea. Because of the manner in which neuroblastomas spread relentlessly to all surrounding organs, they present special problems to the surgeon.

Among the liver tumors, which constitute the third most common tumors of infancy and early childhood, hepatoblastoma and hepatocellular carcinoma are the most common. About a third of liver tumors in children are benign, the most frequent among them being hemangiomas, hamartomas and hemangioendotheliomas. Malignant lymphomas in children can seldom be excised and must be treated with radiation or chemotherapy—usually with somewhat better success than in adults.

In the judgment of this reviewer this well prepared text should be an invaluable asset not only to pediatric surgeons, but to *all* pediatricians.—Paul S. Rhoads, M.D., Richmond, Internal Medicine

Pluribus Press has released Legal Guide for Medical Office Managers. Written by Marshall B. Kapp, J.D., M.P.H., the book emphasizes everyday legal questions as well as major legal challenges. Chapters about medical records, informed consent, vicarious liability, confidentiality and privileged information stress the importance of careful attention to detail. It is an effective tool for training new employees and a convenient reference for seasoned managers. 150 pages, soft cover, \$16.95.

CME QUIZ

TO OBTAIN ONE HOUR OF CATEGORY 1 AMA CME CREDIT, answer the following questions by circling the correct answer on the answer sheet below. Complete and clip the application form and mail it to: Indiana University School of Medicine, CME Division, Fesler Hall 224, 1120 South Dr., Indianapolis 46223.

Alcoholism Research

CONTINUED FROM PAGES 663-668

- Orientals who exhibit the alcohol flush reaction have a deficiency of which enzyme:
 - a. Gamma glutamyl transpeptidase
 - b. Microsomal ethanol oxidizing system
 - c. Aldehyde dehydrogenase
 - d. Alcohol dehydrogenase
- The genetic basis for alcohol abuse is supported by all of the following except:
 - a. Studies on the prevalence of alcoholism in adopted out children of alcoholics.
 - b. Electroencephalogram patterns in identical twins ingesting alcohol.
 - Animal studies comparing ethanol effects in alcohol-preferring and nonpreferring rats.
 - d. Alcohol dehydrogenase isoenzyme patterns in alcoholics.
- Biological responses to chronic excessive alcohol ingestion include all of the following except:
 - a. Induction of microsomal drug ox idizing enzymes.
 - b. Development of CNS tolerance to ethanol.
 - c. Increased high density lipoprotein

- eholesterol.
- d. Microcytosis.
- 4. Alcoholic cirrhosis:
 - a. Occurs in greater than 40% of chronic alcoholics.
 - Accounts for over 100,000 alcohol related deaths per year in the United States.
 - c. Develops after 5-10 years of alcohol consumption in excess of the equivalent of 10 oz. of distilled spirits per day.
 - d. Is a nutritional deficiency disorder.
- 5. Characteristics of Antabuse therapy include all of the following except:
 - a. It is contraindicated in patients with severe cardiac, pulmonary or hepatic disease.
 - Pharmacologically ineffective doses seem as effective as standard dose therapy.
 - c. It can exacerbate depression, and underlying psychosis.
 - d. It is superior to Alcoholics Anonymous in achieving abstinence.
- 6. Which of the following is specific for the diagnosis of alcoholism?
 - a. Transaminase elevation

- b. Macrocytosis
- c. Gamma glutamyl transpeptidase elevation
- d. None of the above
- 7. Which of the following is consistently decreased in the brains of alcohol preferring rats?
 - a. Dopamine
 - b. Serotonin
 - c. Glyeine
 - d. Acetylcholine
- 8. Which of the following has been shown to effectively curtail alcohol consumption in alcohol-preferring rats?
 - a. Fluoxetine
 - b. Imipramine
 - c. Levadopa
 - d. Chlordiazepoxide
- 9. All of the following are strong in dicators of alcoholism except:
 - a. Blood alcohol level greater than 300 mg/dl.
 - Blood alcohol level of 150 mg/dl in a patient who is not obviously intoxicated.
 - c. Blood alcohol level of 50 mg/dl dur ing a routine examination.
 - d. Blood alcohol level of 120 mg/dl during routine examination.
- 10. Accumulation of which substance is felt to be responsible for the clinical syndrome of flushing, vasodilation, headache, nausea and hypotension when alcohol is ingested:
 - a. Acetaldehyde
 - b. Acetate
 - c. Serotonin
 - d. Histamine

JULY CME QUIZ Answers

Following are the answers to the CME quiz that appeared in the July 1985 issue: "Prolonged Apnea in Infancy: Evaluation and Management," by Peter H. Scott, M.D.

M.D.	
1. c	6. e
2. a	7. e
3. d	8. e
4. b	9. a
5. a	10. e

Answer sheet for Quiz: (Alcoholism Research . . .)

 1. a b c d
 6. a b c d

 2. a b c d
 7. a b c d

 3. a b c d
 8. a b c d

 4. a b c d
 9. a b c d

 5. a b c d
 10. a b c d

I wish to apply for one hour of category 1 AMA Continuing Medical Education credit through the I.U. School of Medicine. I have read the article and answered the quiz on the answer sheet above. I understand that my answer sheet will be graded confidentially, at no cost to me, and that notification of my successful completion of the quiz (80% of the questions answered correctly) will be directed to me for my application for the Physician's Recognition Award of the American Medical Association. I also understand that if I do not answer 80% of the questions correctly, I will not be advised of my score but the answers will be published in the next issue of Indiana Medicine.

Name (please print or type)

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For the Asking . . .

- Austin Pathology Associates of Austin, Texas, is making available the rapid fluorescing foci inhibition test (REFIT) for use in determining the immune status of post-exposure patients and high-risk groups for rabies. The occurrence of several instances in which less than effective immune reaction was created by modern methods makes the determination of immune status of great importance. The address is 711 W. 38th St., Suite C-11, P.O. Box 9806, Austin, Tex. 78766.
- · "Drugs-Use, Misuse, Abuse: Guidance for Families" is the subject of Public Affairs Pamphlet 515A. It was written by Margaret Hill, who explores generational differences in drug use. Since everyone does not take to drugs, what is it that influences teenagers and adults, alike, to become addicted? A summary is provided to list personal qualities and experiences that can make a person susceptible. Also covered is a discussion of factors in family life that can act as deterrents. \$1 per copy. Write Public Affairs Committee, 381 Park Ave., South, New York, N.Y. 10016.
- "Anorexia Nervosa and Bulimia: Two Severe Eating Disorders" is the title of Public Affairs Pamphlet 632. The author, Beverly Jacobson, summarizes the psychological profiles of those afflicted by the diseases and lists a variety of approaches to treatment

including a combination of hospitaliza tion, medication (especially for those who are depressed), and psychotherapy for patient and family. \$1 per copy, with discounts for large quantities. Same address as above.

• BNA Communications has a new catalog that features more than 300 films, videos, packaged training programs and training products. It is the most comprehensive collection of state-of-the-art training materials in the world, and is available free of charge. The address is 9439 Key West Ave., Rockville, Md. 20850—(301) 948-0540.

The Stop-a-Stain Pad

A new type of protective pad for use in beds in the home or hospital is now available. It is called the Stop-a-Stain pad and consists of two layers of flannelette bonded to a tough vinyl sheet.

The Stop-a-Stain pad is comfortable in winter and summer and protects the mattress without fail. It is washable, bleachable and tumble dries quickly. The pad, more comfortable than the old-style rubber sheet, is made in sizes for baby carriages, cribs, twin, full-size, queen and king-sized beds. It features strong anchor bands to loop around the the corners of a mattress.

For more information, contact Pillow Talk, Inc., 388 Pond Road, Freehold, N.J. 07728-(201) 780-9483.

Bristol Joins Network of ADD-Vantage Providers

Bristol Laboratories has become the tenth major drug manufacturer to agree to participate in Abbott Laboratories' ADD-Vantage system, which simplifies the intravenous administration of drugs especially packaged for such use.

Besides Bristol, Eli Lilly, Burroughs Wellcome, Hoffmann LaRoche, Upjohn, Wyeth Laboratories, Beecham Laboratories, Miles Pharmaceuticals, Hoechst-Roussel and Abbott's Pharmaceutical Division cooperate in the ADD-Vantage system.

Blood Pressure Research Brings Pleasant Surprise

Jay Zimmerman and fellow Ball State University researchers, in studying the efficacy of various treatment modes on volunteers with a history of hypertension, told the control group that they could lower their blood pressure by taking it regularly. Three other groups were assigned to techniques of meditation, abdominal breathing, and hand and foot warming, respectively.

Blood pressures and the need for medication were reduced in the three relaxation groups and, as a surprise for the researchers, the control group also enjoyed the same results.

Physician Recognition Awards -



The following ISMA physicians are recent recipients of the AMA's Physician Recognition Award. This award is official documentation of Continuing Medical Education hours earned, and is acceptable proof in most states requiring CME in re-registration that the mandatory hours of CME have been accomplished.



Adkins, Stanley R., Columbus Buehl, Frederick H., Vincennes Cline, Donald L., Indianapolis DuBois, Don R., Indianapolis Elek, Kenneth E., South Bend Ellis, Robert F., Merrillville Feuer, Henry, Indianapolis Ford, Thomas E., Valparaiso Gilliland, John E., Franklin Granger, William J., IV, Lawrenceburg

Gruszynski, Thomas R., Granger Hirsch, Herman L., Mount Vernon Jentz, David L., South Bend Judge, Robert E., Fort Wayne Kinasiewicz, Leon E., Crown Point Nero, Richard P., Madison Nowzaradan, Philip, Valparaiso Rusher, Merrill W., Fort Wayne Snodgrass, Robert E., Indianapolis Wolf, Harry C., Indianapolis

New ISMA Members

The following physicians were welcomed in May as new members of the Indiana State Medical Association:

Asok C. Antony, M.D., Indianapolis, internal medicine.

Veena B. Antony, M.D., Indianapolis, pulmonary diseases.

Byron E. Batteiger, M.D., In dianapolis, infectious diseases.

Richard J. Biggerstaff, M.D., Indianapolis, otorhinolaryngology.

John R. Black, M.D., Indianapolis, infectious diseases.

Susanne Blix, M.D., Indianapolis, psychiatry.

Howard S. Boswell, Jr., M.D., Indianapolis, internal medicine.

John D. Bradley, M.D., Indianapolis, rheumatology.

Kenneth D. Brandt, M.D., Indianapolis, rheumatology.

Thomas C. Buchanan, M.D., Terre Haute, orthopedic surgery.

William C. Buffie, M.D., Indianapolis, internal medicine.

Douglas K. Bullington, M.D., Franklin, family practice.

Mary L. Bush, M.D., Indianapolis, obstetrics and gynecology.

Virginia A. Caine, M.D., Indianapolis, internal medicine.

Eric L. DeWeese, M.D., Indianapolis, pulmonary diseases.

Robert S. Dittus, M.D., Indianapolis, internal medicine.

Lawrence H. Einhorn, M.D., Indianapolis, hematology.

Gregory H. Ellis, M.D., Anderson, pathology.

Kenneth H. Fife, M.D., Indianapolis, internal medicine.

Rose S. Fife, M.D., Indianapolis, rheumatology.

S. Edwin Fineberg, M.D., Indianapolis, diabetes.

Douglas R. Flint, M.D., Indianapolis, family practice.

Theodore G. Gabig, M.D., Indianapolis, hematology.

Gabra S. Gachaw, M.D., Indianapolis, psychiatry.

James O. Gates, M.D., Fort Wayne, therapeutic radiology.

Gareth H. Gilkey, M.D., Indianapolis, internal medicine.

Irene M. Gordon, M.D., West Lafayette, therapeutic radiology.

Mary C. Greenlee, M.D., In dianapolis, endocrinology.

Anne Greist, M.D., Indianapolis, oneology.

David D. Hall, D.O., Indianapolis, psychiatry.

James B. Hamaker, M.D., in dianapolis, internal medicine.

Susan E. Hartman, M.D., In dianapolis, family medicine.

David R. Hathaway, M.D., In dianapolis, internal medicine.

Bernard M. Herbst, M.D., In dianapolis, family practice.

Johnny L. Hobbs, M.D., Indianapolis, anesthesiology.

Ronald Hoffman, M.D., Indianapolis, internal medicine.

Daniel J. Hurley, M.D., Indianapolis, internal medicine.

Jan Jansen, M.D., Indianapolis, hematology.

C. Conrad Johnston, Jr., M.D., Indianapolis, endocrinology.

Robert B. Jones, M.D., Indianapolis, infectious diseases.

Walter E. Judson, M.D., Indianapolis, cardiovascular diseases.

Apostolos E. Kalovidouris, M.D., Indianapolis, rheumatology.

Charles R. Kelley, M.D., Indianapolis, internal medicine.

Timothy J. Kelly, M.D., Indianapolis, internal medicine.

Richard B. Kohler, M.D., Indianapolis, infectious diseases.

Michael Kovacich, M.D., Merrillville, family practice.

James C. Liang, M.D., Munster, ophthalmology.

Thomas C. Lloyd, Jr., M.D., Indianapolis, pulmonary diseases.

Patrick J. Loehrer, M.D., Indianapolis, oncology.

Keith W. Logie, M.D., Indianapolis, oncology.

Andrew F. Louden, M.D., Indianapolis, anesthesiology.

James K. Malone, M.D., Indianapolis, diabetes.

Joseph J. Mamlin, M.D., Indianapolis, internal medicine.

Praveen N. Mathur, M.D., Indianapolis, internal medicine.

Clement J. McDonald, M.D., Indianapolis, internal medicine.

Roland B. McGrath, M.D., In dianapolis, internal medicine.

Christopher Melin, M.D., Anderson, neurology.

Howard M. Mishoulam, M.D., Munster, internal medicine.

Stephen L. Myers, M.D., In dianapolis, rheumatology.

Jane A. Pardieck, M.D., Indianapolis, pediatrics.

Don L. Perkins, M.D., Evansvitle, family practice.

David Pletzer, M.D., Noblesville, family practice.

John H. Pratt, M.D., Indianapolis, endocrinology.

Gerald R Rightmyer, M.D., Evansville, family practice.

George W. Sledge, Jr., M.D., Indianapolis, oncology.

David M. Smith, M.D., Indianapolis, internal medicine.

Cheryle D. Southern, M.D., Indianapolis, internal medicine.

Glenn H. Speckman, M.D., Indianapolis, general practice.

Thomas Y. Sullivan, M.D., Indianapolis, pulmonary diseases.

Mark D. Tasch, M.D., Indianapolis, anesthesiology.

Mervin D. Terrell, M.D., Richmond, family practice.

William M. Tierney, M.D., Indianapolis, internal medicine.

Randall M. Todd, M.D., Indianapolis, emergency medicine.

Cathi E. Weatherly, M.D., Indianapolis, RMS.

Myron H. Weinberger, M.D., Indianapolis, endocrinology.

Lawrence J. Wheat, M.D., Indianapolis, infectious diseases.

Arthur C. White, M.D., Indianapolis, infectious diseases.

Stephen D. Williams, M.D., Indianapolis, oncology.

Wesley B. Wong, M.D., Indianapolis, oncology.

Louis M. Wright, M.D., Muncie, internal medicine.

Ibrahim G. Zabaneh, M.D., Valparaiso, family practice.

news notes

Here and There . . .

Dr. Alan T. Marty of Evansville has been elected a founding member of the Society for Neurovascular Surgery.

Dr. James B. Steichen of Indianapolis is a member of the faculty that will teach and discuss "Clinical Reconstructive Upper Extremity Microsurgery" at the Hotel Meridien in San Francisco Oct. 10-12, under the sponsorship of the Ralph K. Davies Medical Center.

Dr. Paul Schoon of Danville has been elected to fellowship in the American College of Obstetricians and Gynecologists.



Dr. Corcoran

Dr. Patrick J. V. Corcoran of Evansville, medical education director at Welborn Baptist Hospital, was honored in June by the Institute for Drug and Alcohol Studies at the University of Evansville; he was cited for his leadership and action in convincing the medical community of the seriousness of alcoholism and the need for treatment.

Dr. Alan D. Schmetzer of Indianapolis has been elected a fellow of the American Psychiatric Association.

Dr. Donald S. Chamberlain of Mishawaka has been elected chairman of the board, American Physicians Life.

Dr. Dean Maglinte of Indianapolis is co-author of the chapter on Conven-

tional Radiology of the Biliary Tract, which appears in the 4th edition of Bockus' *Gastroenterology* (1985, W. B. Saunders Co., Philadelphia).

Lee C. Murphy, Ed.D., formerly an administrator with the nation's preeminent institution for the hearing impaired, Gallaudet College in Washington, D.C., is the new superintendent of the Indiana School for the Deaf.

Dr. Kenneth J. Ahler of Rensselaer has been re-elected president of the Sagamore Council, Boy Scouts of America.

Dr. Larry D. Lovall of Danville discussed drug, alcohol and tobacco abuse with sixth graders at Lincoln Elementary School, Brownsburg, in May.

Dr. John C. Huus is the new medical staff president of Welborn Baptist Hospital, Evansville; Dr. James W. Renne is president-elect, Dr. William C. Wooten is secretary, and Dr. Milan D. Gerlanc is treasurer.

Dr. Randall A. Lee of Martinsville discussed cholesterol in June during a community education program sponsored by Morgan County Memorial Hospital.

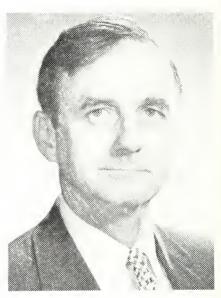
Dr. Bruce A. Lockwitz, an Elkhart rheumatologist, discussed arthritis during a program in May sponsored by Holy Cross Parkview Hospital, Plymouth, and the Arthritis Foundation.

Dr. Zaka-Ur Rahman of Jeffersonville discussed cardiovascular diseases at a recent meeting of the Clarksville Optimist Club.

Dr. John A. Knote of Lafayette has been elected president-elect of the 300-member Organization of State Medical Society Presidents (OSMAP).

Dr. Ara K. Yeretsian of Merrillville, medical director of The Alcoholism Institute of The Methodist Hospitals, discussed "Depression" during a recent community awareness seminar sponsored by the institute.

Dr. Peter C. Kamperschroer of Seymour was the guest speaker at a recent Families Facing Cancer meeting at Jackson County Hospital. Dr. Eric S. Williams of Indianapolis, president of the Marion County Chapter, American Heart Assn., has been appointed to the editorial board of ACCEL, the American College of Cardiology's monthly cardiology audiocassette "journal;" ACCEL, available as a monthly subscription service, features postgraduate programs ranging from lectures and round-table discussions to tapes of ACC scientific sessions.



Dr. Bonsett

Dr. Charles A. Bonsett of Indianapolis, long-time author of INDIANA MEDICINE's "Medical Museum Notes," was presented a recognition plaque by the board of directors of the Indiana Medical History Museum during I.U. Alumni Day this spring; he was cited for his efforts in "preserving Indiana's medical history."

Dr. Cherryl Friedman of Noblesville addressed the Riverview Hospital Cancer Patient Support Group in June.

Dr. Marc E. Weinbaum of Anderson served as moderator at a recent forum on teen suicide sponsored by Anderson's Community Hospital.

Dr. B. D. Patel of Marion discussed lung cancer treatments at a June meeting of Marion's Better Breathers Club

Dr. Duane A. Hougendobler of Huntington has been elected Huntington County health officer.

Helpful Mailing Tips

Pitney Bowes, a mail equipment company, offers several mailing suggestions that could save money, time and paperwork:

- Make addresses machine-readable for faster electronic processing. Addresses that are typed in capital letters without punctuation and that include ZIP codes and two-letter state abbreviations can be "read" by the electronic mail sorters now being used by the U.S. Postal Service.
- Mail early in the day. By avoiding the 5 p.m. rush at the post office, when most business mail arrives, your mail can get on its way faster.
- Use standard-size envelopes. To be processed electronically, envelopes must meet certain size requirements. Odd-shaped or under- and over-sized envelopes have to be hand-sorted and there's a 10-cent surcharge for every one, too.
- Combine items. Instead of separately mailing a bill and an appointment reminder for 44 cents, com-

bine them both in one envelope and mail them for 22 cents.

• Save time with postage-by-phone. Instead of taking your postage meter to the post office to reset, make a 90-second, toll-free telephone call and reset it right in your office. More than half of the postage meter systems being sold today are postage-by-phone systems.

Koala Centers Open New Facility in Fort Wayne

Koala Centers, Indiana's leading hospitals in alcohol and drug abuse treatment, opened a Koala Center in St. Joseph's Hospital, Fort Wayne, July 1. It has 24 beds.

Patients are admitted for a 30-day stay that begins with a medically supervised detoxification program that includes a medical history, physical examination, alcohol/drug evaluation, psycho-social inventory and individualized treatment planning.

The Koala Center staff works close-

ly with the St. Joseph's Hospital medical staff in treating alcoholics and drug abusers, says Harold J. Thompson, Koala vice-president of operations, Indianapolis.

"We believe in a personalized treat ment plan for each patient," Thompson said. "As at our facilities in Lebanon and Columbus, we have a close-knit team of physicians, psychologists, nurses, social workers, certified alcoholism and drug abuse counselors, chaplain, recreation therapist and dietitian."

Calendar Reminder: ISMA Convention

The ISMA's annual convention will be conducted at the Century Center in South Bend, Wednesday through Sunday, Nov. 13-17.

Hotel accommodations for the convention will be in the South Bend Mariott Hotel. Watch your mail for convention registration information.

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OBITUARIES

Thomas O. Dorrance, M.D.

Dr. Dorrance, 75, a retired Bluffton pediatrician, died June 20 at Caylor Nickel Hospital.

He was a 1936 graduate of Rush Medical College, Chicago, and was an Army veteran of World War II. He retired in 1975.

Dr. Dorranee had been associated with the Caylor-Nickel Medical Center since 1937. He was a former Wells County health officer. He was a diplomate of the American Board of Pediatrics and a member of the American Academy of Pediatrics and the American College of Physicians.

Francis G. Zeier, M.D.

Dr. Zeier, 74, an orthopedic surgeon associated with the Welborn Clinic in Evansville until he retired earlier this year, died June 7 at Welborn Baptist Hospital.

He was a 1936 graduate of Northwestern University Medical School, Chicago, and was an Army veteran of World War II.

Dr. Zeier, who served as a professor of orthopedics on the hospital ship SS Hope from 1963 to 1971, was a fellow of the American Academy of Orthopaedic Surgeons. He was a diplomate of the American Board of Orthopaedic Surgery.

James W. Denny, M.D.

Dr. Denny, 85, a retired Indianapolis general practitioner, died June 16 at his home.

He was a 1923 graduate of Indiana University School of Medicine and was a veteran of World War I.

Dr. Denny, who retired from practice in 1982, was a past president of Methodist Hospital, Indianapolis. He was vice-president of the first board of trustees at Community Hospital. He was a member of the ISMA Fifty Year Club.

Rex M. Joseph, M.D.

Dr. Joseph, 67, a retired Beech Grove general practitioner, died June 26 at his home in Danville.

He was a 1944 graduate of Indiana University School of Medicine. He retired in 1980 after serving six years as clinical director of the family practice residency training program for St. Francis Hospital Center, Beech Grove.

Dr. Joseph, a past president of the medical staffs for St. Francis Hospital Center and University Heights Hospital, was an assistant professor of clinical medicine in the Dept. of Family Practice, I.U. School of Medicine. He was a diplomate of the American Board of Family Practice and a member of both the American Academy and the Indiana Academy of Family Physicians.

Nathaniel D. Ewing, M.D.

Dr. Ewing, 68, a retired Vincennes surgeon, died June 8 at Good Samaritan Hospital.

He was a 1943 graduate of Washington University School of Medicine, St. Louis. He served in the Army from 1945 to 1947.

Dr. Ewing, who retired in 1980, was a former president of the Knox County Medical Society. He was a member of the International College of Surgeons and the American College of Surgeons. He founded the Good Samaritan Hospital Cancer Clinic in 1977 and served as its director until his retirement.

Glen W. Cartwright, M.D.

Dr. Cartwright, 48, a pediatrician at the Arnett Clinic in Lafayette, died June 4.

He was a 1961 graduate of Indiana University School of Medicine and served in the Air Force from 1962 to 1964

Dr. Cartwright was a member of the American Academy of Pediatrics and was certified in neonatology by the American Board of Pediatrics.

J. Leon Simms, M.D.

Dr. Simms, 71, an Indianapolis internist, died June 15 at his home.

He was a 1945 graduate of Meharry Medical College, Nashville, Tenn., and was a veteran of World War II and the Korean War.

Dr. Simms, a staff member of Winona, Methodist and St. Vincent Hospitals, was a member of the American Academy of Family Physicians.

Memorials: Indiana Medical Foundation

The Indiana Medical Foundation, Inc. was formed by the Indiana State Medical Association "for religious, charitable, scientific, literary or educational purposes." It provides financial assistance to support the educational mission of Indiana Medicine.

Contributions made to the Foundation are deductible by donors in accordance with the Internal Revenue Code. Gifts are deductible for Federal estate and gift tax purposes.

The Foundation is pleased to acknowledge the receipt of gifts in remembrance of the following individuals:

Edwin W. Dyar, M.D. Maurice E. Glock, M.D. Guy A. Owsley, M.D. Wilbert Smith Eugene S. Rifner, M.D. Elsie A. Reid Lester D. Bibler, M.D. Lłoyd A. Vogel, M.D. Arvine G. Popplewell, M.D. Mildred Ramsey

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AMA ALL DELEGATES (Ferms end Dec. 31)

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- Pres Gordon I. Gutmann, JeHersonville Secy Olegario J. Ignacio, lettersonville Annual Meeting 1986, leffersonville
- 4-- Pres. Rosemary E. Wen, Brownstown Secy Charles Calhoun, Seymour Annual Meeting, May 7, 1986, Seymour
- 5- Pres Michael S. McCrea, Terre Haute Secv. Peggy Sankey Swaim, Rockville Annual Meeting: Sept. 1985, Brazil-
- 6- Pres Dean Felker, Greenfield Secv. Douglas Morrell, Rushville Annual Meeting; Sept. 12, 1985, Rushville
- T-Pres John M. Records, Franklin Secv. Marshall H. Trusler, Indianapolis
- Annual Meeting 8-Pres: Conrado R. Miranda III, Winchester Secy. Jerome M. Leahey, Umon City Annual Meeting: June 4, 1986, Delaware
- 9-Pres: Walter P. Beaver, Noblesville Secy. Dennis L. Pippenger, Noblesville Annual Meeting: June 11, 1986, Noblesville
- 10- Pres. Robert J. Bills, Merrillville Secy Barron M. Palmer, Hammond Annual Meeting, Oct. 9, 1985, Hobart
- H-Prest Phil O. Burgan, Kokomo Secy: Fred Poehler, Lal-ontaine Annual Meeting: Sept. 18, 1985, Kokomo
- 12 =Pres: Antonio B. Donesa, Fort Wayne Secy Mark S. Souder, Auburn Annual Meeting, Sept. 19, 1985, Fort Wayne
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